The Journal

OF THE KANSAS MEDICAL SOCIETY

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1. Wilhelmj, C. M.; McDonough, J., and McCarthy, H. H.: Nutrition and Blood Pressure, Am. J. Digest. Dis. *20*:117 (May) 1953.
2. Mann, G. V., and Stare, F. J.: Nutritional Needs in Illness and Disease, J.A.M.A. *142*:409 (Feb. 11) 1950.
3. McLester, J. S., and Darby, W. J.: Nutrition and Diets in Health and Disease, ed. 6, Philadelphia, W. B. Saunders Company, 1952, pp. 519-524.
4. Levine, V. E.: The Blood Pressure of the Eskimo, Federation Proc. *1*:121 (Mar. 16) 1942.

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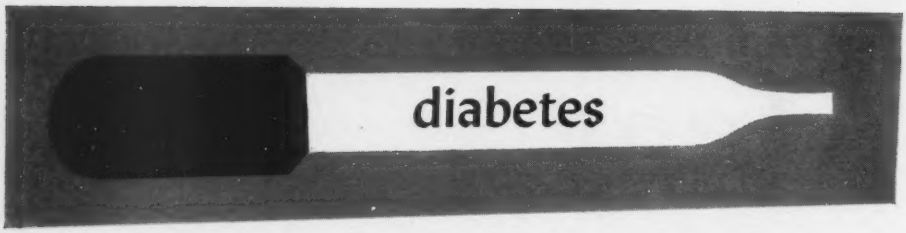

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1. Blotner, H., and Marble, A.: New England J. Med. *245*:567 (Oct. 11) 1951.
2. Steine, L.: GP *8*:45 (July) 1953.

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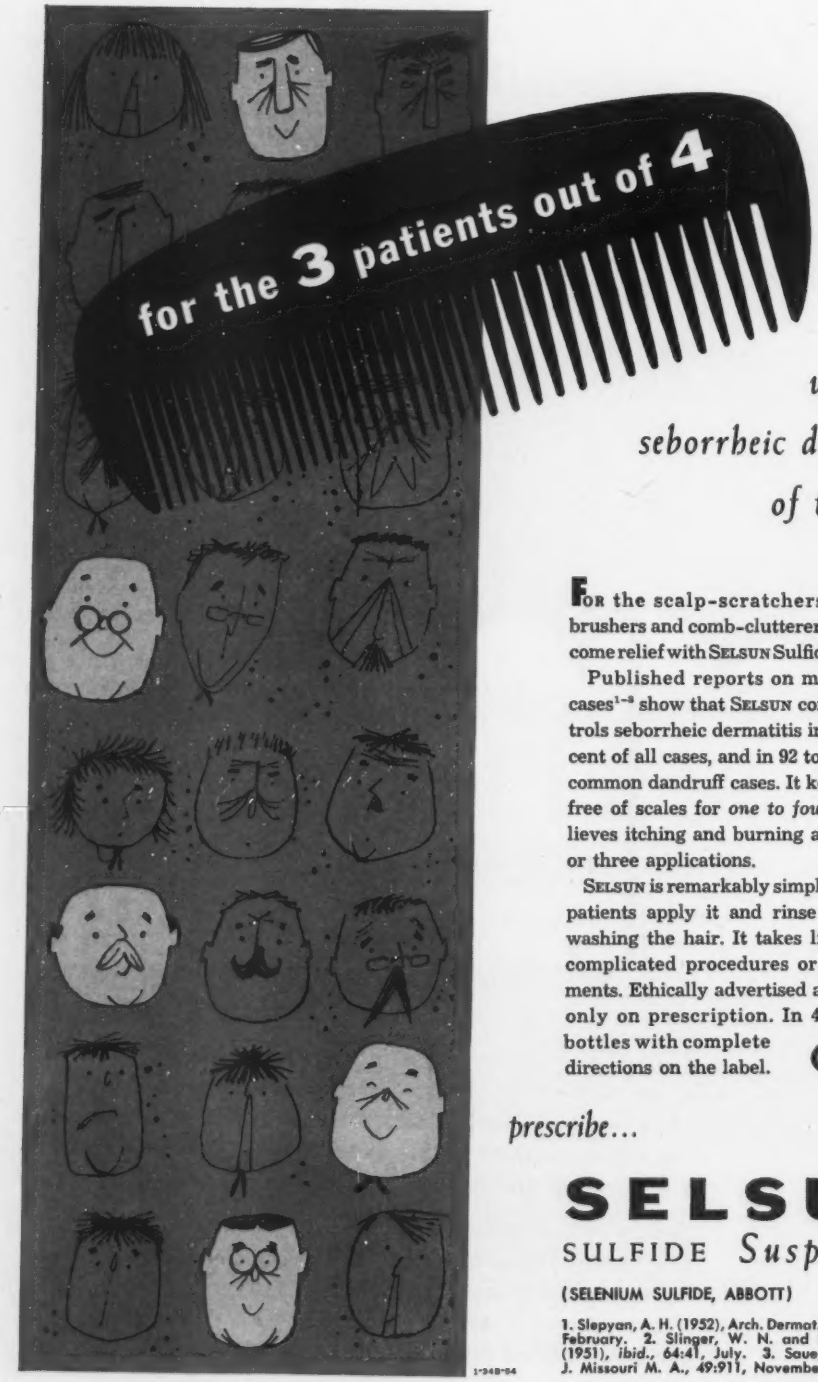

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1. Slepyan, A. H. (1952), Arch. Dermat. & Syph., 65:228, February. 2. Slinger, W. N. and Hubbard, D. M. (1951), *ibid.*, 64:41, July. 3. Sauer, G. C. (1952), J. Missouri M. A., 49:911, November.

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1. Kauntze, R., and Trounce, J.: Treatment of Arterial Hypertension with Veriloid (Veratrum Viride), Lancet *2*:1002 (Dec. 1) 1951.

2. Wilkins, R. W.: Combination of Drugs in the Treatment of Essential Hypertension, Mississippi Doctor *30*:359 (Apr.) 1953.

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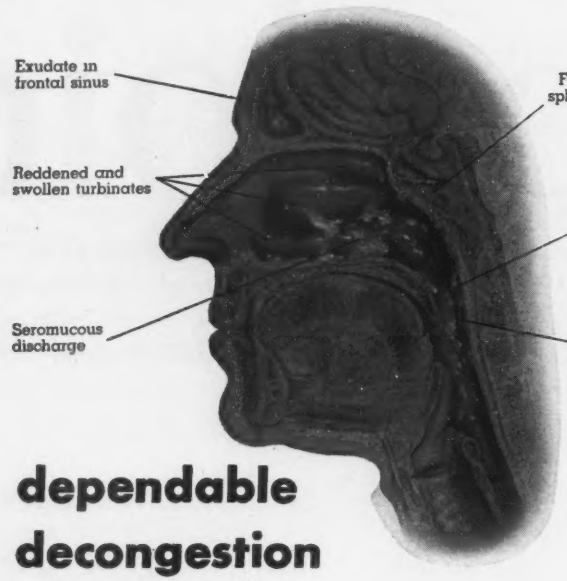

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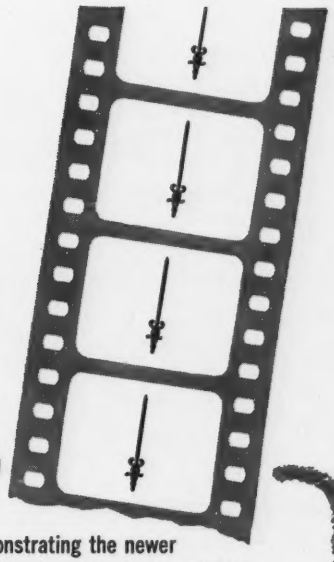

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Volume LV JANUARY, 1954 No. 1

The Treatment of Diabetic Acidosis and Diabetic Coma

Edward Tolstoi, M.D.
New York, New York

There is no one accepted technique for the treatment of diabetic acidosis and coma at present. Therefore, I can only discuss the method I have used and with which I have had the greatest experience. It is not presented as the most perfect plan of treatment, but as a plan that has given us satisfactory results. It is simple, safe, effective and does not require a specialist. No doubt there are many other methods, differing in details though not much in principle, which might have been just as efficacious in our hands. But, since I have had insufficient experience with other techniques, I shall describe ours in detail, and in passing make some comment about other methods.

For clarity I shall present the subject under the following headings: 1. Definition; 2. Presentation of cases and their management; 3. Why ketoacidosis develops; 4. How ketoacidosis develops; 5. A discussion of the accepted and controversial principles and procedures of treatment.

1. DEFINITION

A. *Diabetic Acidosis:* This is a clinical picture in which a patient with diabetes develops increased thirst, frequency, weakness, acetone breath, nausea, vomiting, abdominal and chest pains, soft eye balls, as well as other evidences of dehydration and air hunger (Kussmal breathing). The laboratory studies will reveal a glycosuria, ketonuria, hyperglycemia, leukocytosis and a low carbon dioxide combining power. However, the patient is conscious.

B. *Diabetic Coma:* When a patient with diabetic acidosis loses consciousness, then and only then is the diagnosis of diabetic coma made. Such a patient may be semi-conscious; that is, he will respond to stimuli, but may be unable to take fluids or answer questions, or, he may be unconscious so that he does not even respond to stimuli. I agree with Owens[1] that the term diabetic coma should be used only for those patients in ketosis whose mental state has become one of unconsciousness. It is wholly a clinical diagnosis. True, the carbon dioxide combining power is usually low. This indicates the degree of the acidosis only, but certainly not the mental status of the patient.

2. PRESENTATION OF CASES AND THEIR MANAGEMENT

A. *The Mild Case.* Often a trained diabetic patient under observation will carry on, feel well, hold his weight, and be free from symptoms, and though he has a glycosuria he will not show acetone in the urine. At times, during a slight upper respiratory infection, a gastrointestinal upset, or an extreme sunburn, or for no apparent reason, his thirst will increase, the urinary output rises, and acetone is found in the urine. The qualitative reading of the reaction may be 1 to 4 plus. Such a patient is not hospitalized. He is usually familiar with the tests for acetone and sugar and can follow his treatment at home. He is then told to follow these directions:

From the Department of Medicine, New York Hospital and Cornell University Medical College, New York City.
Associate Professor of Clinical Medicine, Cornell University Medical College; Attending Physician and Chief, Diabetic Clinic, New York Hospital; Consulting Physician, New York Infirmary for Women and Children, and Norwalk Hospital, Norwalk, Connecticut.

1. Test your specimens, every time you void, for sugar and acetone.

2. If acetone is present, take regular insulin in amounts depending upon the quantity of sugar found in the urine. That is, if the test if yellow, brown or orange (4 plus) take 25 units: if the test is green with yellow sediment, take 15 units: if the urine is blue or sugar free, take orange juice.

3. Drink all you can—water, milk, fruit juices, ginger ale.

4. Take salty broths as often as desired.

5. Continue this routine until 3 consecutive specimens are acetone free. If acetone persists and you feel nauseous or vomit, come to the hospital at once.

By means of this home treatment the ketosis clears rapidly and as the underlying cause improves, the patient can resume his former regimen. In those in whom the ketosis appears without apparent cause, I have learned that an increase of their daily dose of insulin remedied the situation.

B. *The Moderately Severe and Severe Case.* Before presenting a description of this group, I want to state categorically that every newly diagnosed diabetic with ketosis, regardless of its severity, should be hospitalized, chiefly because of lack of training. However, I must state that because of hospital overcrowding I have treated some of these cases at home. Here the help of a nurse or a member of the family was urgently needed. I taught them the routine of testing, left definite written instructions, kept in touch by telephone at hourly intervals, and made calls as often as I could. There were no dire consequences.

Now, the moderately severe case developing ketoacidosis and coma may reveal this pattern. The patient develops a sore throat or some other infection. His temperature rises, he develops malaise, and he has no desire for food. Since he decided not to eat, he sees no necessity for taking insulin as he fears a reaction. He may not eat for a day, two days or more, subsisting on fruit juices and other fluids and still reasoning, though fallaciously, that insulin under these circumstances is unnecessary. Soon he observes an increase in thirst and notes some frequency. He then develops aching legs, weakness, restlessness, and, perhaps, some somnolence. He carries on. Soon there appear epigastric pain, nausea, and vomiting. His heart beats rapidly, and it becomes difficult for him to breathe. He or some member of the family becomes alarmed, and either a physician is called or the patient is taken to the hospital. Often no medical aid is sought until the patient is or has been unconscious for many hours. When such a patient is admitted to the hospital, the infection will be treated by any of the modern chemotherapeutic or antibiotic agents, and his diabetic acidosis, coma, or both will be treated as follows:

1. If the patient is unconscious, insert a catheter into the bladder and clamp it. This will facilitate getting samples of urine for analysis.

2. Give 25 units of regular or crystalline insulin subcutaneously every half hour until the clinical symptoms of ketosis begin to disappear and the urine becomes acetone free.

3. If the patient can take and retain fluids, give 2 or 3 glassfuls (200 cc.) of fruit juice every half hour to one hour. Salty broths and other liquids are given as frequently as the patient desires.

4. If fluids cannot be taken by mouth, give 1000 cc. of 5 per cent glucose in saline at once, rapidly. The infusion is then continued at a slower rate until the oral route can be used. In connection with any

TABLE 1

		BLOOD		URINE			
TIME	INSULIN	CO_2	SUGAR	SUGAR	ACETONE	DIACETIC	
8:00	25 units	19	325	4 plus	4 plus	4 plus	1000 cc 10% G. in Sal.
8:30	25 "			4 "	4 "	4 "	400 cc water
9:00	25 "			4 "	4 "	4 "	200 cc O.J.
9:30	25 "			4 "	4 "	4 "	500 cc N/6 lactate
10:00	25 "			4 "	4 "	2 "	200 cc water
10:30	25 "			4 "	4 "	0	200 cc water
11:00	25 "			4 "	4 "	0	1000 cc 10% G. in Sal.
11:30	25 "			4 "	4 "	0	400 cc water
12:00	25 "			4 "	4 "	0	200 cc water
12:30	25 "			4 "	4 "	0	200 cc O.J.
1:00	25 "			4 "	4 "	0	400 cc water
1:30	25 "			4 "	4 "	0	400 cc water
2:00	25 "			4 "	4 "	0	1000 cc 10% G. in Sal.
2:30	25 "			4 "	4 "	0	100 cc O.J.
3:00	25 "			4 "	3 "	0	200 cc water
3:30	25 "			4 "	3 "	0	200 cc water
4:00	25 "			4 "	2 "	0	100 cc grape J.
5:00	25 "			4 "	±	0	500 cc 10% G. in Sal.
6:00	25 "			4 "	0	0	200 cc water
7:00	25 "			4 "	0	0	

Patient had diabetes for 12 years. Was taking R20—0—P25. Well until one month ago when he began losing weight; polyuria and loss of appetite. Three days ago was nauseated and did not take insulin. For 2 days before admission vomited all food, became drowsy, and, when admitted, vomited a small quantity of blood.

P.E. P120 R40 BP 105/50 T37
Semi-stuporous, Kussmal, acetone in breath, eye balls soft. Pharynx red.
HBO 16: RBC 5.1 : WBC 29,800: BUN 86
Acetone free in 9 hours.

continuous intravenous therapy, great care must be exercised to prevent overloading the right heart. This hazard is present in the older diabetic with arteriosclerosis and the young cardiac.

5. After the urine has become free from acetone bodies continue giving 25 units of insulin and 2 glasses of orange juice every two hours for four or more doses.

Of 165 cases of diabetic acidosis and coma, so treated, there were 19 deaths of which only 6 were due to the acidosis per se, and of these one was highly insulin resistant.

3. WHY DOES KETOACIDOSIS DEVELOP?

The factors responsible for the development of diabetic acidosis and coma are: 1. failure to take insulin; 2. infection; 3. combination of the two. In Joslin's experience, "The commonest cause of diabetic acidosis continues to be laxity of treatment, consisting of dietary indiscretions or neglect to take an adequate amount of insulin, or both." These factors were the cause of 53 per cent of a group of cases, infection being accountable for only 23 per cent. In my experience, infection and omission of insulin were the commonest causes for the precipitation of ketoacidosis and coma.

The dietary breaks were not as impressive, and after an analysis of 27 of our patients who did not adhere to a diet, we found that only 2 were eating more while 25 ate less than their prescription called for. In a group of 220 instances of diabetic acidosis and coma, Beardwood and Rouse[2] state, "Only nine can be listed as breaking diet." Though there is a difference of opinion concerning the "breaking of diet" as a factor in the development of diabetic ketoacidosis, it can be categorically stated that the most important cause is the omission of insulin, or a relative insufficiency of it, as during an infection its efficacy is considerably reduced. Consequently, a unitage which may have been sufficient during health to maintain the urine free from ketones, becomes inadequate during an infection, and ketosis ensues.

4. HOW KETOACIDOSIS DEVELOPS

At this point it is of interest to inquire how the diabetic develops ketoacidosis and coma. As you know, the overabundance of acetone bodies, acetone, diacetic, and beta hydroxbyteric acids, in the blood and tissues causes an acidosis. This is brought about by a depletion of base. It was once thought that these acetone bodies were abnormal and products of fat metabolism. This hypothesis is not tenable today.

The experimental studies of many investigators have established the following facts:

A. Ketone bodies are normal intermediates of fatty acid catabolism in the liver—the only organ which manufactures them in significant quantities. B. Ketone bodies so made in the liver are secreted into the blood stream where they are utilized by the peripheral tissues. This occurs in both normal and diabetic subjects, whether or not insulin or carbohydrate is available. C. Ketone bodies will not cause acidosis as long as their production does not exceed their utilization. However, when they are produced at a greater rate than they can be utilized by muscle and other tissues, they must of necessity accumulate, and it is this accumulation in the blood and tissues, due to over-production, that starts the vicious cycle of acidosis, depletion of base, dehydration, and coma.

The next query is, "Why does the liver overproduce these ketone bodies?" The obvious reply is that the liver is using for its energy the substance from which these bodies are derived—namely, fatty acids. Stadie's[3] experimental studies led him to the conclusion that when the demand for fat calories exceeds about 2.5 gms. per kilogram per day, the production of ketones exceeds utilization resulting in ketonuria, and, if unchecked, leads to ketoacidosis and coma.

How can we prevent the liver from subsisting on fatty acids? By making carbohydrate available as liver glycogen. Liver glycogen has been termed the greatest factor of safety against ketogenesis. Here the hypothesis has been proposed by Mirsky[4] and supported by fine experimental evidence that a decrease of the liver glycogen is the most likely stimulus to ketone formation. This is quite logical, for, as the available carbohydrate is lowered, the liver will metabolize protein and fat. And as the combustion of fat reaches the figure of 2.5 gms. per kilogram per day, as postulated by Stadie, the production of ketones will be accelerated to a point surpassing utilization with a sequential ketosis.

If, therefore, we can offer the liver carbohydrate (glycogen) instead of fat, will ketogenesis be decreased? Evidence answers this query rather conclusively. A rapid drop in ketonemia and ketonuria was observed by Mirsky[4] in depancreatized dogs following the administration of glucose intravenously. Root and Carpenter[5] also reported a drop in blood ketones from 70 to 44 mg. in a diabetic patient 3 hours after 50 gms. of glucose were given by vein. It is obvious that glucose alone can decrease acetone formation in both the diabetic animal and the diabetic patient, but, to suppress ketogenesis, insulin is also needed. It has been shown that the glycogen content of the diabetic liver increases more markedly when insulin is also used, and that the glucose so stored is not so rapidly dissipated, thus offering the liver carbohydrate rather than fat for its own metabolic needs.

It is obvious from what I have said that the diabetic patient will develop ketosis and coma if the liver catabolizes fatty acids in abnormal quantities.

This the liver will do if glycogen is not available for its energy requirements. And, in the diabetic the reduced glycogen stores are further lowered by omission of insulin, infection, or any other condition which reduces the potency of insulin.

5. PRINCIPLES AND PROCEDURES OF TREATMENT

Each group employing the technique it developed for the treatment of diabetic acidosis insists that all steps of treatment are based on sound physiologic grounds. Mortality statistics are produced to support the thesis that such treatment is most satisfactory, and, as additional support explanations are offered, why the death rate is higher elsewhere. Such comparative data are neither satisfactory nor valid because of the multiplicity of factors which determine the prognosis in each case.

It is generally agreed that the results are dependent on: A. the duration of unconsciousness; B. the age of the patient; C. the presence of serious medical complications.

If similar cases could be treated by different methods, the superiority of one method over another could be determined. However, such an ideally controlled clinical experiment is always beyond hope. Owens[1] has stated that even the so-called "best therapy" in the unconscious diabetic is highly unsatisfactory while the duration of acidosis without unconsciousness is of considerably less importance. All agree that for best results early treatment is imperative.

The most commonly and frequently employed measures in the treatment of diabetic acidosis and coma are:

1. insulin
2. fluids and salt
3. blood analysis
4. glucose
5. potassium
6. adjuvants
   a. N/6 molar lactate
   b. transfusions
   c. alkalis
   d. lavage
   e. enemas
   f. stimulants

Everyone agrees that insulin is most urgently needed as diabetic acidosis and coma are the end results of a relative or absolute insulin insufficiency. Everyone, therefore, prescribes insulin—the difference is a quantitative one. The dosages recommended by various workers have varied from 25 to 400 units as the initial dose. Whenever the smaller doses are given, they are repeated at one-half to hourly intervals until improvement as judged by clinical and laboratory data is apparent. Olmstead[6] gives 100 units at once, and, if there is no drop in the blood sugar after 1½ hours, another 100 units are given. Joslin's group of 141 cases received an average of 206 units during the first three hours.

There are still other variations, and the fact that good to excellent results have been obtained by all methods suggests that insulin is important, but that one dose or its frequency of administration is not superior to another dose given more or less frequently. We have given 25 units, a small dose, at frequent intervals of 30 minutes. This plan, though empirical, has been satisfactory. That does not mean that giving 50, 75, or 100 units is not good therapy, even though the outcome in a series of cases may be poor.

Owens[1] used 50 units of insulin every half hour at the start, but he reports a high mortality. Of 92 patients admitted, 49 were unconscious with an average of 16 hours of coma. Among this group of 49, there was a death rate of 73.5 per cent. Does that mean that Owens' technique is inferior to ours, or that some one bungled the therapy? Not at all. His work is done in a general hospital with an active ambulance service, and he is called upon to treat more severe and more neglected cases than we do.

I decry the statement that some one has blundered if death occurs as a result of diabetic acidosis or coma. Many patients die in spite of generally accepted "superior" therapy, and no satisfactory explanation can be offered as to why death occurred. Schacter, Weisel and Cohn[7] suggest that peripheral vascular failure causes such deaths. Danowski, Winkler and Peters[8] state that the profound loss of salt and water may be the causal factors of such failure. These hypotheses may be tenable as the patient in ketosis with shock has a decreased blood volume, a hemoconcentration, a hypochloremia, a hyperpotassemia, and a falling blood pressure. This may be the picture terminally. However, I have observed comatose diabetics whose blood pressure was maintained, whose chemical picture was corrected, and in whom no clinical evidence of peripheral failure was apparent. The therapy was considered adequate, and yet such patients never regained consciousness.

The use of salt and fluids to replace the loss of base and water is so universal that no comment is needed. Often sixth-molar lactate was also used, and transfusions were given only as indicated. We do not rely upon the level of the blood sugar, nor carbon dioxide combining power, as a guide of treatment. In our hospital and probably in others, the results of the blood examination may not be available for one to three hours, at which time the data are of academic interest. Our principal laboratory guide is the examination of urine for sugar and acetone at ½ hourly intervals, and we continue the insulin dosage of 25 units as long as acetone is present. The test is reliable, rapid and therefore practical.

The question of whether or not glucose should be used in the treatment of diabetic acidosis and coma has been argued with much passion and emotion. Many statements concerning the deleterious effects of glucose have been based on circumstances that were merely fortuitous. Our experience with glucose has not been unfavorable. As I have indicated above, we have used it as a 5 per cent and occasionally as a 10 per cent solution intravenously. The median given in a 24-hour period was 112 grams. When the amount given by mouth was added to that, the median amount rose to a total of 312 grams for the first 24 hours, with a minimum of 50 grams and a maximum of 800 grams.

It has been stated by Root[9] and his associates that glucose in doses of 50 or more grams is harmful because: 1. It obscures the actual blood sugar values, thus eliminating a guide for insulin dosage; 2. It may produce liver damage; 3. It neutralizes the action of insulin; 4. The hyperglycemia thus produced is harmful to the pancreas; 5. It may provoke anuria; 6. Such large doses are not necessary as the body can metabolize only 5 to 10 gms. per hour.

These statements are made by respected leaders in the field with such conviction that the physician whose opportunity to treat diabetic acidosis is limited, accepts them as facts. It is desirable to appraise them and see what evidence is available to support them.

That the administration of glucose obscures the actual blood sugar is a fact, and I have no objection if one wishes to use the blood sugar as a therapeutic guide. I maintain, however, that it is excessive ketonemia and not hyperglycemia which is the underlying cause of diabetic acidosis and coma, and, therefore, ketone bodies in the urine are more accurate guides of therapy. Insulin should be continued frequently until the ketone bodies vanish, and then it is no problem if one wishes to treat the hyperglycemia.

Astwood, Flynn, and Krayer[10] have shown that liver damage resulted in dogs receiving glucose by vein at a rate exceeding 40 gms. per square meter per hour. They also demonstrated that dogs indefinitely tolerated 12 gms. per square meter per hour. These experiments have been offered as evidence that glucose may produce liver damage. Such reasoning is fallacious. First, excessive quantities of any innocuous agent, even water, will prove toxic, and while a dog may not be able to tolerate large quantities of glucose, that does not necessarily indicate that man cannot do so. Recently we had an opportunity to observe a patient in whom large quantities of glucose were not hepatotoxic. Following a transfusion, a young woman developed a kidney shutdown. To facilitate diuresis, large quantities of glucose were given by vein. The data is shown in Table 2.

This patient, as you see, received from 300 to 870 gms. of glucose daily by vein. There was no glycosuria. Most of her blood sugars were normal, and one is led to the conclusion that glucose so given was metabolized at a rate of 12.5 to 36 gms. per hour. Histologically the liver showed no abnormalities. Rabinowitch, Fowler and Bensley[11] have shown that the diabetic with the aid of insulin can also utilize more than 5 to 10 gms. of glucose per hour, as shown in the table.

TABLE 3

HOURS	32	48	43
GLUCOSE INTAKE	1020	780	835
EXCRETED	10	32	63
UTILIZED	1010	748	772
GMS/HOUR	31.5	15.5	18.0

It is true that Root and Carpenter[5] used a different approach in determining how much glucose was metabolized. However, other data as well as ours are clear. It is obvious that if the glucose was not excreted, it must have been used by the body.

The "neutralization" of insulin by glucose is questionable. Perhaps that is not what was meant, but the choice of the word "neutralize" was not the most desirable one.

The interaction of insulin and glucose is not the same as the neutralization of an acid by an alkali. Insulin favors the accumulation of glycogen, but its

TABLE 2

DATE	FLUID INTAKE	URINE OUTPUT	GMS. GLU. 24/HRS.	UREA N.	BLOOD				ICTERIC INDEX
					SUGAR	CO_2	CHLORIDES	SERUM P.	
2/11	6350	170	870	65	223	62	479	4.4	5
2/12	2700	73	490	65	133	60	469	5.5	—
2/13	3795	21	760	69	190	49	446	5.5	11
2/14	2360	30	300	73	85	57	429	5.5	
2/15	3930	47	700	85	89	51	423	5.1	
2/16	3900	37	400	—	—	—	—	—	
2/17	2800	105	300	—	—	—	—	—	
2/18	4970	108	425	89	77	45	399	4.8	5
2/19	3815	908	400	100	79	41	376	—	
2/20	3885	1930	300	104	—	40	385	5.0	
2/21	4330	3150	500	111	85	40	396	5.1	
2/22	4200	3850	500	102		45	421	—	
2/23	0	4200	400	96		37	424	5.4	

mode of action is not yet understood. Its action has been compared to an enzyme subtrate reaction, and, whether or not an excess of glucose is present, a certain amount will be acted upon by the quantity of insulin available. The beneficial effect of the insulin is not nullified by excessive amounts of glucose.

The harmfulness of excessive quantities of glucose to the pancreas, in the treatment of diabetic acidosis and coma, is highly speculative. My own observations on postmortem material failed to reveal histological changes in the pancreas. This is not an unusual observation as Root also quotes two fatal cases which were treated with glucose in addition to insulin and whose blood sugars were 475 and 667 mgm. per cent, and yet the autopsies were negative. It seems to me that there is altogether too much condemnation of glucose in the treatment of diabetic acidosis and coma. This indictment is not based on sound evidence.

I doubt whether the use or non-use of glucose is the crucial issue. I am sure that suitable cases will recover both with and without the use of glucose as long as adequate doses of insulin, salt, and fluids are used. The case in which there has been a long period of unconsciousness will probably die in spite of the so-called very best treatment. Personally, as I stated, with frequent small doses of insulin and the use of glucose, I have found the results good. The addition of glucose gives an added feeling of security that I will not throw my patients into the hypoglycemic phase, thus putting an additional burden upon a circulatory system already under considerable stress.

Potassium. In 1946 Holler[12] noted that a patient, while recovering from diabetic coma, developed paralysis and at the same time exhibited a low serum potassium. The administration of a 2 per cent solution of potassium chloride intravenously resulted in a dramatic recovery from the paralysis. It was quite natural, therefore, that potassium salts began to be used rather freely and generally in the treatment of diabetic acidosis and coma. As experience with potassium salts as a component of treatment increased, many physicians urged that their use be most judicious because of toxic reactions. This is not the place to enter into a detailed discussion of potassium metabolism. Briefly, it is accepted by those who have had experience with potassium, that the greatest danger of a hypopotassemia is during the recovery phase, after the patient's acetone has cleared because of large doses of insulin, fluids, salt, and glucose. At this stage the patient is usually conscious, not nauseous, and is able to retain fluids. Consequently, the safest and best way to replenish the lost potassium content is the administration of substances rich in potassium. Orange juice, grapefruit, and grape juice are ideal. If more potassium is desired, it can be administered as the chloride or citrate as 1.0 gm. every 6 hours.

However, if after diligent and adequate therapy, the patient is still unconscious and one wishes to administer potassium, a solution can be prepared from commercially available ampules containing 1.5 gms. per 10 cc. Usually such a 10 cc. ampule is added to 500 cc. of 5 per cent glucose and permitted to flow into a vein at the rate of 60 to 75 drops per minute.

Prior to the intravenous use of potassium, one must ascertain that there is a hypopotassemia and that the kidneys are functioning well with a good urinary output. Furthermore, the serum potassium levels should be determined by the flame photometer and also electrocardiograms. While the electrocardiographic tracing usually reflects the serum potassium concentration, at times the correlation is poor. I have not used potassium routinely by vein as it is a hazard. It should be employed sparingly, judiciously, and only by those whose facilities for analyses are good and whose experience has been extensive. The oral route is safer and certainly less troublesome.

SUMMARY

1. Patient with diabetic acidosis or diabetic coma can be successfully treated with small and frequently repeated doses of regular insulin—25 units every half hour—replacement of water, salt, and glucose by mouth, vein, or both.

2. The administration of glucose by vein or orally is beneficial and not harmful. Data are presented to support this thesis.

3. Potassium salts are not used routinely in the treatment of diabetic ketosis and coma. This does not mean that they have no place in therapy. They have a definite value, but there are hazards associated with their use. Both of these points have been discussed.

4. It is unnecessary to follow the blood sugar and the carbon dioxide combining power as guides of therapy. Our experience has shown that clinical response and disappearance of ketone bodies are excellent guides.

5. The earlier the treatment is begun, the more favorable is the prognosis, and, exclusive of medical complications, it can be stated categorically that the longer the patient remains unconscious, the less favorable are his chances for recovery.

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Management of Acute Laryngotracheobronchitis

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In a recent symposium at a national meeting, croup and laryngotracheobronchitis were listed under the same title, although I believe the intent was to give consideration to the latter disease. Croup is a relatively benign disease, and its management should prove no problem. Confusion of the two conditions may be due to the fact that acute infective laryngotracheobronchitis has sometimes been called non-membranous croup to distinguish it from diphtheria, formerly known as membranous croup.

The condition under consideration may be defined according to Jackson[1] as "an acute inflammation of the upper and lower air passages with specific clinical features but not always attributable to the same infective agent." The disease in its incipiency is frequently mistaken for croup, and it may be confused with laryngeal diphtheria, but its fulminating character and severity should change this opinion; resultant deaths, often diagnosed as pneumonia or bronchopneumonia, are in reality the terminal phases of unrecognized acute laryngotracheobronchitis.

To set the stage for our discussion, I should like to point out a few etiologic and diagnostic features because I so firmly believe that they have an important bearing on treatment. The chief predisposing cause is age, the greatest incidence being infancy to four years, with the highest mortality under two years. Factors contributing to the clinical picture in relation to age are: (1) Lack of adequate immunity in a child who has never had an acute respiratory infection. (2) Anatomic reasons: in the early years of life, there is much more loose areolar tissue in the larynx, especially in the conus elasticus. The triangular aperture of the infant larynx[4] is 7 mm. in anteroposterior direction by 4 mm. in width across the posterior commissure. The glottic chink has an area of approximately 14 square mm. An edema of 1 mm. of the mucosal surfaces of this glottic chink will reduce the airway to 5 square mm. Also the lumen of the airway below the larynx is relatively small. Thus, an exudate 1 mm. in thickness on the lining wall will completely occlude a 2 mm. bronchus while the same exudate in a 4 mm. bronchus would leave a 2 mm. lumen. (3) Physiologic factors: the bechic blast (force of cough) in a baby is about 1/10 that of an adult, and the child is unable to expel sticky secretions from small tubes. The cough reflex may be obtunded or absent because of toxemia or ill advised drugs.

In addition to the above, a careful history will commonly reveal contact infections of the influenzal type with tracheobronchitis, in older members of the family. I can personally testify to a reverse twist of this situation when, in 1936, I treated a serious case of laryngotracheobronchitis which required bronchoscopy and tracheotomy. As a result, in 48 hours I contracted a severe tracheobronchitis which progressed to bronchopneumonia. As an adult I survived, but the child succumbed.

The hairline distinction between obstructive non-diphtheritic croup and infective laryngotracheobronchitis is not important; both usually present the same characteristics in typical cases: toxemia, edema of the larynx, and thick viscid obstructive, often crusting, non-membranous exudate in the tracheobronchial tree.

Gilbert[2] has given us a useful classification according to the pathologic changes present and often observable with the laryngoscope or bronchoscope:

1. Acute catarrhal laryngotracheitis in which the laryngeal and upper tracheal mucosa is reddened and velvety, secretions are mucoid and easily aspirated, and the cords freely movable; clinical symptoms of hoarseness, brassy cough, and obstruction are comparatively mild.

2. Supraglottic edematous obstructive laryngitis in which the epiglottis is enormously swollen and beefy red, edema involves the aryepiglottic folds and arytenoids so that one or both cords are fixed. The picture of obstructive dyspnea is rapidly progressive. The voice is guttural, wavering, and described as having a "hot potato" quality. The patient assumes an up-

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right position with chin forward and mouth open, and respiratory distress increases in the recumbent position.

3. Subglottic obstructive laryngitis and tracheitis further subdivided into (a) the exudative or inspissated type, and (b) the edematous type. In the exudative type, laryngoscopic inspection reveals vocal cords slightly separated by moist exudate or crusts; after aspiration the cords move freely. A congested nonedematous mucosa is seen in the subglottic area, and dyspnea is relieved, if only temporarily. In the edematous type, supraglottic structures are normal, the cords are slightly mobile or fixed in the midline, and suction does not increase their mobility; the subglottic edema narrows the airway and prevents normal abduction of the true cords during inspiration. Hence, aspiration of secretions does not relieve the dyspnea in this type.

4. Acute obstructive laryngotracheobronchitis, according to Gilbert[2] is a term applicable to only a small group of cases (8 per cent in his series of 2,550 cases) in which subglottic edematous laryngitis with fixed cords is combined with edematous tracheobronchitis and a mucopurulent exudate or gummy material which extends into the smallest bronchial radicles. Aspiration, tracheotomy, or even bronchoscopy may not relieve these patients who are usually under 1 or 2 years of age, and the mortality rate remains about 70 per cent. In my opinion, it is in this type of patient that the recent combination of a wetting agent as advised by Gorman,[3] along with cold humidification, offers some additional hope by liquefaction of the almost solid exudate and making possible catheter aspiration through the tracheotomy tube.

As with all classifications, this one has its limitations; various types may not be sharply differentiated or may merge one with the other. Any intelligent consideration of this disease entity adds up to the inevitable conclusion that we are dealing with a serious illness in the younger age group.

In medical management of the disease, the young patient requires prompt judgment when first seen. The notoriously rapid onset, stridulous croupy respirations, high fever, and extreme toxicity give warning, but obstruction to breathing decides that hospitalization must be immediate.

The most important therapeutic indication is placement of the patient in a room saturated with moisture. Steam is the traditional method and in some cases is effective. However, the steamy room with temperature at 90°, the ceiling and walls dripping, a constantly moist bed, are overbearing and can be harmful. A nurse is able to work under such conditions only a few hours. The same situation within a crib-tent embarrasses the patient. The larger the tent, the easier the handling, and the patient has the feeling of adequate space for breathing.

Cool moist air is soothing to the mucous membranes and helps to lower body temperature and metabolic rate; hence, there is minimum oxygen consumption. Loss of body fluids is also much less.

Relative humidity of 70 to 90 per cent with oxygen concentration of 40 per cent at a room temperature of 65° to 75° can be maintained by various methods. With infants, a Burgess box serving as a croup tent into which moisture combined with a wetting agent is nebulized, is quite satisfactory. The wetting agent, sodium lauryl sulfate (Duponel) is cheap and nontoxic, and humidity can be noticeably increased, up to 30 per cent, by its use. Oxygen from a cylinder at the rate of 4 to 6 litres per minute can be used as the nebulizing agent. Several other means have been devised, but our judgment favors the Walton centrifugal humidifier, which is safe, durable, and requires no mechanical tricks to operate. It can be used in conjunction with the oxygen tent if desired.

General measures for the comfort and nutrition of the patient should be so arranged to cause minimum disturbance. Postural drainage as advocated by Galloway[10] is of great advantage. Adequate fluid intake is essential; it should be encouraged by mouth when possible and supplemented parenterally if needed. Since restlessness is an important diagnostic sign of increasing obstruction, sedatives should not be used. Opiates and atropine are strongly contraindicated.

Early antibacterial treatment has been a potent factor in the decrease of morbidity. Culture and sensitivity studies would be desirable if time permitted, but the urgent need for active treatment precludes such study in most cases. Hemolytic streptococcus and staphylococcus and hemophilus influenzae are the most constant causative organisms.

Penicillin becomes the first choice of antibiotics, and it must be given in full dosage. Streptomycin, because of its selective effect on influenzal infections, may be combined with penicillin. Aureomycin, terramycin, and the sulfonamides may have value in some cases.

Before discussing the actual surgical procedures available for treatment, it should be useful to outline some of the indications for surgery. The medical management, as outlined, may obviate the necessity for surgical intervention. The important indications then would seem to be:

1. Failure to improve under medical care with watchful and alert observation.

2. Increase in the signs of obstructive dyspnea, such as stridorous breathing, air hunger, retraction at the suprasternal notch and epigastric (xyphoid) region, also supraclavicular, infraclavicular, and in-

tercostal retraction. True cyanosis is a late symptom, and to wait for its appearance will be too late. Pale cyanosis described as an ashen gray color is an important sign of serious obstruction.

3. Restlessness as exhibited by a sudden start on awaking when a worn out child has momentarily lapsed into fitful sleep and suddenly discovers that his airway is obstructed.

4. Lessening or disappearance of cough because the reflex has been obtunded by toxemia or the use of sedative or mucosal drying drugs.

5. Sudden hyperpyrexia is an unfavorable diagnostic and prognostic sign.

Having decided that surgical care is needed, what are the possible methods available? Direct laryngoscopy may be used for augmenting the diagnosis, for aspiration of secretions and crusts, but is most important as a preliminary to introduction of the bronchoscope for deeper aspiration and to provide an immediate airway. Hollinger and Johnston[4] have pointed out that instrumentation in an infant's larynx may precipitate complete respiratory obstruction by laryngospasm incident to introduction of the laryngoscope. One must therefore be prepared to establish an airway by Mosher life-saver, intratracheal catheter, or bronchoscope. This opinion is shared by Everett[9] who states, "It is difficult to imagine a completely harmless laryngoscopy."

Formerly intubation was used either by the indirect O'Dwyer method or the direct method of Mosher, but this was probably a carry-over from the days of widely prevalent laryngeal diphtheria. It has been largely discarded because the presence of an intubation tube requires the constant attendance of a physician capable of immediately replacing a coughed out or occluded tube, and it permits no aspiration of secretion from the trachea and lower respiratory tract. Repeated bronchoscopy is not advisable as it encourages laryngeal edema and usually gives only transient help.

The operation of choice in the acutely obstructed patient is tracheotomy,[5] and while a competent trained laryngologist should be familiar with the technique, complications, and aftercare of tracheotomy, there may be occasions when those less skilled are called upon to perform the operation.

When tracheotomy is anticipated as an orderly procedure, as much preparation as possible should be made in advance so that unnecessary confusion will not add to the apprehension of a dyspneic patient. Instruments should be sterilized, suction machine available, anesthetic syringes, solutions, etc., ready for use. Position of the patient is important, with a sandbag under the shoulders (not the head or neck) so that the head is extended; the chin must be in the midline. Extremities and the body must be carefully restrained before draping. Introduction of a Mosher life-saver or small bronchoscope facilitates aspiration, maintenance of an airway, and oxygen insufflation. Local anesthesia is introduced by a succession of intradermal wheals in the midline from the suprasternal to the thyroid notch. A few deeper injections may be made in the midline but are not necessary laterally as the operation is advisedly limited to a midline procedure. Jackson[1] forbids general anesthesia under any circumstances, while a few authors, including Crowe,[6] sanction light ether anesthesia with oxygen through a bronchoscope or intratracheal catheter, thereby making the operation much less of a shock or struggle for an ill child. This can be discontinued as soon as the trachea is opened.

Midline incision through the skin and superficial tissues is made from the Adam's apple to the suprasternal notch, taking care not to cut through the perichondrium of the laryngeal cartilages. Hemostasis is secured to make a dry wound, as in any surgical operation, and the incision is deepened to the pretracheal fascia, identified by the ridges of the tracheal rings felt by the finger. The thyroid isthmus, if atrophic, may be retracted upward or downward; if sizable, it should be incised between two clamps introduced from above down and ligated lateral to the clamps. After exposing the trachea, a hook tenaculum is used to fix and slightly lift the trachea by passing it below and under the second ring in the midline.

Vertical incision of the cartilages of the 3rd and 4th rings is now made, with care that the knife does not go deep enough to cut the posterior tracheoesophageal party wall. Priest[7] injects a few drops of 5 per cent cocaine solution into the trachea a few moments before opening, but Jackson[1] objects to this procedure as the cocaine dulls the cough reflex which acts to expel secretions on opening the trachea. Removal of a semicircular section from each side of the vertical incision leaves a circular opening about the same size as the tracheotomy tube and may avoid pressure necrosis at a later time.

The edges of the incision are spread with a Trousseau dilator to allow catheter suction in the trachea and easy introduction of the proper size tracheotomy tube with obturator; the latter is commonly forced out by bechic blast on release, but, if not, it should be removed and replaced by the inner tracheotomic canula. The upper end of the wound may be closed by two sutures well away from the tube, if desired, but leaving the lower end of the wound unsutured makes less likely the unhappy complication of emphysema in the tissues of the neck or even in the mediastinum. This will be discussed more fully later.

A split gauze dressing placed under the shield

and around the canula of the tracheotomy tube can be replaced when soiled and encourages normal healing of the wound in the presence of serum from the tissues. The tapes from the two sides of the shield should be joined around the neck and tied in a square knot rather than a bow knot. Failure to fix the tube snugly in position may allow a tube to be extruded, and deaths have resulted from this avoidable accident.

Emergency tracheotomy may occasionally be imperative. A knife and a pair of trained hands are enough,[1] but full surgical equipment is better. No anesthesia is needed for a patient requiring a life-saving emergency tracheotomy. The Mosher life-saver, which can be introduced by touch and used in a home, may obviate the emergency and make possible an orderly operation. The same cardinal principles of incision in the midline, feeling for the tracheal rings, and opening of the trachea with a finger limited scalpel, govern the emergency operation.

In the aftercare of tracheotomy patients, it is trite to say that success depends largely upon good nursing. However, the surgeon must not relinquish his responsibility or supervision to see that the proper care is forthcoming. Since the patient has been temporarily deprived of voice and ability to cry, he must be watched day and night lest the newly provided airway become again obstructed. Moisture must be supplied, preferably by a mechanical cold vaporizer, so that room or crib temperature is not above 72°F. In Europe[4] during World War II, a significant reduction in cases of laryngotracheobronchitis was observed when fuel was scarce and houses were heatless during most of the winter months. Before the war, this infection was prevalent in the winter months. Transportation of a sick child from the heated surroundings at home through the cool night air to the hospital has shown a similar favorable effect.

As previously noted, under medical care the addition of a wetting agent, sodium lauryl sulfate (Duponel C), as reported by Gorman,[3] appears to be an advantage. One cc. dissolved in one gallon of water can raise the relative humidity of a small room from 65 to 90 within 30 minutes. The same agent in .1 per cent solution in warm normal saline may be instilled directly into the tracheotomy tube, followed by gentle catheter suction to assist in removing the dry secretions and crusts from the tracheobronchial tree. The inner tracheal canula should be changed frequently by nurses, and the outer tube should be inspected daily by the surgeon and changed as needed. A Trousseau dilator and duplicate tracheotomy tube should be at the bedside; in some cases it may become necessary to remove crusts by passing a small bronchoscope through the tracheotomy wound. The patient must be watched for signs of hypoxia.

Decanulation (removal of the tracheotomy tube) can usually be accomplished in 7 to 10 days, but this should not be attempted until the patient has been on trial for 24 to 48 hours with the tube corked. Since many of these small patients become panicky when the tube is removed, having learned to depend upon it for free breathing, it may be wise to substitute the next smaller size tracheotomy tube for 2 or 3 days before attempting decanulation. If the tracheotomy tube has been placed at the 3rd or 4th ring, decanulation is not usually difficult. On the contrary, an emergency "stab" operation performed near the cricoid may result in stenosis, a troublesome complication.

Tracheotomy may be subject to some serious or even fatal complications. Emphysema, by forcing of air through the tracheotomy wound into the fascial planes of the neck and thence to the mediastinum, should be suspected when hypoxia and dyspnea recur in the presence of a clear airway. This accident is favored by suturing the wound, especially the lower end, too close to the tube. Hollinger[8a] has described another mechanism. He states that mediastinal emphysema and pneumothorax following tracheotomy with a bronchoscope in place are due to too low a tracheotomy, caused by hyperextension of the head which draws the trachea out of the thorax.

The fact that serious complications can and do occur is attested by Everett[9] in an analysis of acute laryngotracheobronchitis cases, 1940 to 1949. In a group of 1,077 patients, tracheotomies were performed in 86 (8 per cent) with a mortality of 11.6 per cent. Pneumothorax resulted in 10.4 per cent of the tracheotomized cases; there were two bilateral cases, both fatal, and 7 unilateral cases with one death. In the unilateral cases, 6 out of 7 occurred on the left side. This latter coincides with the reports of other observers. Simpson[8] compares the former average of about 50 per cent mortality with more favorable results in recent years, incident to the use of the new drugs and earlier tracheotomy, but he notes that in a group of 64 cases requiring tracheotomy, 12 were fatal (18 per cent). Bronchial obstruction was the most frequent cause of death.

SUMMARY AND CONCLUSIONS

1. Acute laryngotracheobronchitis is a fulminating medical and surgical emergency because of the danger of obstruction to breathing. The rather sudden onset, high fever, stridor, and extreme toxicity distinguish the infection from croup. Etiological factors are discussed; the condition can be rapidly fatal; serious signs and symptoms are given.

2. Early recognition, hospitalization, and proper medical treatment decrease the need for surgical in-

tervention. Cool humidity by various means is discussed and is favored. Oxygen concentrations of 40 to 50 per cent are necessary. The use of antibiotics is outlined with reasons for the choice of penicillin, streptomycin, and aureomycin; other medical measures are mentioned.

3. The surgical treatment of acute infective laryngotracheobronchitis becomes a question of good judgment and weighing the factors: (1) the degree of airway obstruction, and (2) the ability of the circulatory system to stand the overload. Tracheotomy is the procedure of choice, and it should be performed relatively early rather than as a last resort. The indications for operation, technique, aftercare, and complications have been described and the results of treatment have been discussed.

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The Diagnosis of Lead Poisoning

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INTRODUCTION

Lead was one of the first metals used by man. Lead intoxication or lead poisoning, the terms being synonymous, was known to the Greek, Roman, and Arabian physicians long before the time of Christ.[9]

Today, lead poisoning is rarely seen outside of the "lead industries." The non-occupational exposures are the ones we all heard of as medical students, being presented to us as someone's diagnostic accomplishment. Typical of these case reports was the comatose child who was found to have eaten leaded paint from his crib and the woman with colic who had burned discarded storage batteries. Some of these sporadic poisonings are listed in Table 1.

TABLE 1

CAUSES OF NON-INDUSTRIAL LEAD POISONING

1. Burning storage batteries.	
2. Infants chewing paint from cribs.	
3. Lead in rubber nipples and toys.	
4. Amateur painting at home.	
5. Snuff wrapped in lead foil.	
6. Wines and beer made at home.	
7. Galvanized nails held in the mouth.	
8. Drinking of "canned heat."	
9. Handling of lead seals.	
10. White lead used as a fingerprinting powder.	

These poisonings are rare now, thanks to careful public health measures. In this regard, it is probably news to most of us that lead poisoning is a reportable disease in Kansas.

By far the greatest lead poisoning problem today is encountered in industries. Lead poisoning is second only to dermatoses in the frequency with which it causes disability of workers. Several hundred occupations have been called the "lead industries." A few of these are listed in Table 2.

TABLE 2

WORKERS EXPOSED TO LEAD

1. Storage battery assemblers	11. Ink makers
2. Auto "stream liners"	12. Rubber workers
3. Painters	13. Insecticide manufacturers
4. Welders	14. Tetraethyl lead workers
5. Lead smelters	15. Plumbers
6. Pottery makers	16. Blacksmiths
7. Enamelers	17. Air craft workers
8. Printers	18. Gardeners
9. Solderers	19. Tree sprayers
10. Brass workers	20. Canners

Of these, the storage battery plants account for the greatest number of lead poisoning casualties. In all of these industries, the workers are known to be exposed to lead and are assumed to have absorbed a certain amount of lead. The diagnosis in these workers depends on a differentiation between lead absorption, which is nondisabling, and lead poisoning, which is disabling.

PATHOGENESIS

Some confusion exists in the use of the terms acute

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and chronic lead poisoning. I believe with others[5] that so-called "chronic" lead poisoning is a misnomer and such previously labeled states represent in fact a series of bouts of acute poisoning. Cantarow and Trumper[8] in 1944 exhaustively presented the studies leading to our present concept of the pathogenesis of lead poisoning.

Lead products may be inhaled, swallowed, or absorbed through the skin. By far the most important means of entry is the respiratory tract. Most lead compounds can be demonstrated in the tissues of experimental animals a few hours after intratracheal instillation. Finer particles of lead are assimilated most rapidly, supposedly by phagocytosis; however, volatile lead products, particularly tetraethyl lead, may be absorbed directly from the skin or lungs. Swallowed lead is many times less toxic than inhaled lead, 84 per cent of swallowed lead being excreted in the feces.

Regardless of the portal of entry, lead is distributed throughout the viscera immediately after entering the blood stream. The liver, muscles, and spleen are the usual sites of early deposition, an exception being tetraethyl lead which is readily soluble in fats and concentrates in the central nervous system. A few days after absorption, lead is found in ever-increasing quantities in the bones where it is stored as the phosphate, regardless of the compound which was originally absorbed. There is a parallelism between the behavior of calcium and of lead. The factors which favor the deposition of calcium also favor the deposition of lead, and the same applies to mobilization. Stored lead in the bones is not disabling, but it may be released during subsequent illnesses and then cause symptoms. The human body can accommodate large quantities of lead, when gradually absorbed, without producing symptoms. This state of being "in equilibrium" with lead is spoken of as lead absorption and is clinically important only as it may be a forerunner of lead poisoning. When absorbed lead causes symptoms, it is assumed that organ damage has occurred. Lead poisoning most significantly affects the hematopoietic system, the muscles, and the nervous system (Table 3).

TABLE 3

PATHOGENESIS OF LEAD POISONING

Hematopoietic System
RBC—hemolysis, stippling
Hgb—faulty synthesis
WBC—lymphocyte shift
Smooth Muscle—direct effect
Voluntary Muscle—phosphocreatine metab.
Central Nervous System—
Brain—ganglion cells, capillary damage, exudation, hypertensive damage (?)
Meninges—irritation
Spinal Cord—ant. horn cells degen.
Other Systems Involved—vascular tree (?), kidneys (?), joints (?), gums

I. *Hematopoietic System:*

a. Red blood cells:

Lead causes an increased hemolysis of the red blood cells, and in consequence anemia occurs.

Increased basophilic stippling of the red cells occurs. This is a nonspecific reaction of the red cells which may follow a number of stimuli as listed in Table 4.

TABLE 4

CAUSES OF "STIPPLING"

Infectious disease—e.g. pneumonia
Blood diseases—anemia, leukemia, Hodgkin's disease
Advanced malignancy
Poisoning—lead, mercury, arsenic, aniline, nitrobenzol, carbon disulfide, benzol, gasoline, phenyl-hydrazine
Cirrhosis of the liver

Statistically, however, stippling is most often due to lead. The granules in the cells are not precipitated lead, but are of cytoplasmic origin. Usually stippled cells disappear from the blood two to four weeks after cessation of exposure to lead.

b. Hemoglobin:

Lead interferes with the synthesis of hemoglobin,[17] lowering the hemoglobin level and causing an organic pigment normally contained in the heme component of hemoglobin to be excreted as coproporphyrin III in large amounts in the urine. Most reports, and my experiences, indicate that the urinary porphyrin test is usually an earlier guide to lead damage than is the stipple count.[7, 11, 17] Just as increased stippling is not specific for lead poisoning, neither is an increase in the urinary porphyrin level. Some of the conditions noted to cause this finding are listed in Table 5.

TABLE 5

CAUSES OF PORPHYRINURIA

Infectious dis.—pneumonia, poliomyelitis, syphilis
Liver dis.—hepatitis, cirrhosis
Blood dis.—aplastic anemia, hemolytic anemias, Hodgkin's dis., Addisonian anemia, polycythemia
Deficiency dis.—pellagra, sprue
Skin dis.—eczema, lupus
Occupational exposure—lead, mercury, arsenic, beryllium, selenium, phosphorus, aniline, carbon tetrachloride, benzene
Drugs—alcohol, salicylates, phenacetin, barbiturates

An increase of urinary porphyrins due to stimuli other than lead poisoning is small. Normal urine, for instance, contains 10 to 30 micrograms of porphyrin per liter; in benzene poisoning this level may be 255, while in lead poisoning it is over 3,500 micrograms per liter.[17]

c. White blood cells:

The total white cell count in lead poisoning is

usually normal. Lead damage has been felt to alter the ratio of large mononuclear cells to small lymphocytes.[15]

II. *Smooth Muscle:*

The direct effect of lead on the smooth muscle of the intestinal tract is thought to explain the constriction of the small intestine and the resultant colicy pain which is most characteristic of all the symptoms of lead poisoning. X-ray studies during colic do show hypermotility and spasm of the small intestine with subsequent atonicity as the pain eases.

III. *Voluntary Muscle:*

"Lead palsy" or the muscle weakness seen in lead poisoning is due to a direct effect of lead on the voluntary muscles. Specifically, lead interferes with the resynthesis of phosphocreatine. Because this chemical effect hinders the recuperation of muscles following use, the most exercised muscles, such as the wrist, are involved.

IV. *Central Nervous System:*

a. Brain:

The ganglion cells in the brains of cats are damaged when the cats have been experimentally poisoned with lead. Other studies have indicated that cerebral symptoms may be dependent on capillary damage with subsequent exudation. The cerebral manifestations may mimic hypertensive encephalopathy clinically, and the frequent occurrence of hypertension with this presenting picture has been thought to indicate hypertension, secondary to lead, as the causative factor.

Certainly any of these mechanisms could account for the convulsions and coma which typify cerebral damage in humans.

b. Meninges:

There is irritation of the meninges, as evidenced by increased cerebrospinal fluid pressure, increased protein, and pleocytosis.

c. Spinal cord:

Degeneration of the anterior horn cells has been noted at autopsy, and this may explain the muscle atrophy occasionally seen in lead poisoning.

V. *Other Systems Involved:*

Less frequent and more controversial are the effects of lead on the vascular tree, particularly its association with hypertension.[1] There is no doubt that hypertension occurs frequently in association with lead colic, but this may be purely a reaction to pain. Too, as mentioned before, its more than casual frequency in connection with cerebral symptoms has been thought to indicate that lead causes hypertension and the cerebral symptoms follow. In studies of patients exposed to lead for many years, there is no convincing evidence that hypertension is more frequent than would be expected in an unexposed group.

Damage to the renal tubules may occur in massive lead poisoning, the changes being similar to those produced by other heavy metals. Repeated episodes of poisoning, over a long period, have been thought by some to cause nephrosclerosis as a component of generalized vascular damage. Oliguria, hematuria, and albuminuria may be noted coincident with the appearance of symptoms of lead poisoning, these urinary findings usually clearing as the symptoms are relieved.

Joint pains do occur during lead poisoning, and no morphological changes in the joints have been found to explain this. Stored lead in the bones does not cause these or other symptoms.

Lead combines with decomposed food and gum tissues to form the typical "lead line" in the gums. This consists of a number of minute granules of lead sulfide in the gums. Poor dental hygiene is necessary for its formation, and even with severe lead poisoning the "lead line" may be minimal if the gums are scrupulously clean.[2, 3]

CLINICAL MANIFESTATIONS

TABLE 6

CLINICAL MANIFESTATIONS
History
Symptoms:
Abdominal colic
Muscle weakness
Joint pains, diarrhea, nausea, fatigue, metallic taste
Central nervous system symptoms
Physical signs:
Rigid abdominal wall with colic
Elevated blood pressure with colic
Muscle weakness
Pallor, lead line
Central nervous system signs

I. *History:*

In practice, a history of exposure to lead is often the clue to the diagnosis.

II. *Symptoms:*

By far the most common symptom of lead poisoning is abdominal colic.[4, 5] This may be sudden in onset, but it is often preceded by vague abdominal pains and constipation. The pain is severe and griping and is located in the mid or lower abdomen. Usually each colic lasts several minutes, and the colics recur several times hourly. A sense of pressure may persist between colics. Vomiting is not a prominent feature, though retching is common. The colic is promptly relieved by calcium salts given intravenously.

Muscle weakness, particularly wrist weakness, may

accompany colic, though more often it is a later symptom.

It is interesting that Benjamin Franklin[10] wrote a letter in 1786 which graphically describes these two most important symptoms. He wrote that when he was a boy he heard of a complaint from North Carolina against New England rum. It "poisoned their people, giving them the dry belly-ache, with a loss of the use of their limbs. The distilleries being examined on the occasion, it was found that several of them used leaden stillheads, and the physicians were of the opinion that the mischief was occasioned by that use of lead."

Less frequent and less characteristic symptoms are joint pains, diarrhea, nausea, fatigue, and a metallic taste.

An exception to the usual symptom picture is a predominant, early involvement of the central nervous system. This is now rarely seen, but it does occur in infants, regardless of the lead product involved, in adults poisoned suddenly with massive amounts of any lead product, and in adults poisoned by tetraethyl lead. In each of these instances, the symptoms may be sudden in onset with a rapid progression to convulsions, coma, and death. Insomnia, restlessness, headache, and depression may occasionally constitute a prologue to such central nervous system involvement.

III. *Physical Signs:*

There are very few specific signs of lead poisoning. During bouts of colic, the abdominal muscles are rigid, and the blood pressure is often elevated. The patient evidences distress, often doubling up and pressing his abdomen, which procedure seems to give a measure of relief. Between bouts of colic, the abdomen is doughy and not tender to moderate palpation.

Muscle weakness, particularly of the extensor muscles of the wrists, may be demonstrated by having the patient attempt dorsal extension against resistance. There are no accompanying sensory changes.

Signs of lead absorption, which may be present in the absence of symptoms, but which are almost invariably present during lead poisoning, are pallor and a lead line in the gums. The lead line is seen with a hand lens as a series of minute blue-black particles on the margin of the gums, and not on the teeth.

In the rare instances of central nervous system involvement, the signs may mimic encephalitis in their variability. Convulsions, delirium, blindness, aphasias, and psychoses may come on very rapidly and proceed through coma to death.

LABORATORY FINDINGS

A summary of the laboratory tests altered in lead exposure, absorption, and poisoning is listed in Table 7. An asterisk marks those tests which are most important to the practicing physician.

TABLE 7

ABNORMAL LEAD VALUES

Exposure:	
Air	more than 1.5 mg/10 cu. liters
Absorption:	
Red Cell count*	less than 4.5 mil/cu. mm.
Hemoglobin*	less than 80%
"Stipple" count*	more than 5/50 HPOIF**
Blood lead content	more than .08 mg/100 cc.
Urine lead content	more than .08 mg/liter
Bone x-rays in children	diaphyseal deposition
Damage (?):	
"Stipple" count*	more than 20/50 HPOIF**
Urine porphyrin test*	pink fluorescence

* Easily performed in office laboratory.
** HPOIF—High power oil immersion fields.

I. *Measure of Exposure:*

The measure of lead particles in the air is the best laboratory reflection of the degree of exposure to lead. Actually, this test is valuable in determining the lead hazard in particular industries, but it is outside the means of the practicing physician. It is interesting that less than 1.5 mg/10 cu. liters is not considered an abnormal concentration, implying that we are all exposed to lead "normally," albeit small amounts.

II. *Measures of Absorption:*

The tests altered by lead absorption are the red cell count, the hemoglobin determination, the stipple count, the quantitative blood, the quantitative urine lead tests, and (in children) x-rays of the bones.

a. Red blood cell and hemoglobin determination:

The anemia is hypochromic, though seldom severe. The red cell count is usually less than four and one-half million but rarely less than four million, and the hemoglobin is usually less than 80 per cent, though rarely less than 60 per cent.

b. Stippling of the red blood cells:

Stippling of the red cells is best seen by examining a single layer of cells stained with Wright's stain. They may be tabulated in terms of stippled cells per 50 high power oil immersion fields (abbreviated HPOIF) or as stippled cells per million red cells. Over five stippled cells per 50 fields is evidence of absorption.

c. Quantitative blood lead level:

The concentration of lead in whole blood bears no consistent relationship to the appearance of clinical manifestations.[1, 6] Symptoms may be present at low concentrations or absent at high concentrations.[16] This discrepancy has been suggested to be due to the relative distribution of lead between plasma and red cells[8] and the inability of the commonly used tests to differentiate these fractions. Probably less than

10 per cent of the blood lead is in the plasma, and this fraction is in turn divided into a larger non-dialyzable colloid form and a smaller dialyzable form which is the portion of importance in causing lead damage. Since these divisions of blood lead represent such minute amounts, even in the aggregate, the test aims to determine only the whole blood content.

d. Quantitative urine lead level:

As in the instance of blood lead level, an elevated urine lead level is indicative of increased absorption, transportation, and excretion, and is not diagnostic of lead poisoning.[1, 11, 13] In most cases of frank lead poisoning, the urine lead value is increased, but this may be less than that seen in many instances of simple absorption. The urine content does not bear a constant relation to the blood level, renal function being reflected in the urine determination. The urine test, like the blood test, measures minute quantities of lead, so the contamination by the patient in voiding and collecting the sample is a major factor. The state of hydration of the patient is reflected when the figures are recorded in milligrams per liter.

In both the blood and urine determinations, special lead free glassware must be used, and I feel that these tests are not suited to an office laboratory. The Kansas State Board of Health will make a study of an industrial plant, if requested, and urine and blood determinations may be a part of that study, but the state laboratories do not accept individual samples of blood or urine sent in for lead determinations.[14]

e. X-rays:

X-rays of the growing ends of the bones may show a lead deposit in children who have absorbed much lead, though in adults x-rays are of little help.

III. *Measure of Damage:*

Though the tests for absorption are usually positive in lead poisoning, the tests more specifically indicating damage are excessive stippling of the red cells and a positive urinary porphyrin test.

a. Stipple count:

The stipple count is probably a better guide to lead damage than the blood or urine lead level. Stippling may be absent in the rare cases of sudden massive ingestions of lead with a rapid onset of symptoms, but usually over 20 stipple cells per 50 oil immersion fields are present in lead poisoning, and serial determinations show an increase of stippled cells as poisoning becomes manifest.

b. Semi-quantitative urine porphyrin determination:

The urinary porphyrin test is a simple determination applicable to the average practitioner's laboratory. I use the method of McCord,[12] slightly modified, as illustrated in Table 8.

TABLE 8

URINARY PORPHYRIN DETERMINATION

1. Pipette 10 cc. of a "random" sample of urine into a screw top tube.
2. Add 2 drops of glacial acetic acid.
   Add 2 drops of hydrogen peroxide.
   Add 2 *cc.* of ether.
3. Mix gently in screw top tube.
4. Let stand 30 minutes.
5. View in a dark room using an ultra-violet lamp.
6. Record any pink fluorescence of the surface ring as POSITIVE.

There is no correlation between the quantitative amount of lead in the urine and the presence of a positive porphyrin reaction.[12] The test may vary from positive to negative in the course of a few months time, or it may stay positive long after cessation of exposure to lead when damage supposedly persists.[6, 9]

SUMMARY

Lead poisoning occurs when absorbed lead produces sufficient damage to cause disabling symptoms.

The diagnosis of lead poisoning is based on the history, symptoms, and signs, coupled with the laboratory findings, and is not based on any one specific laboratory test.

TABLE 9

DIAGNOSIS OF LEAD POISONING

History of exposure
- Usual—occupational
- Rare—non-occupational

Symptoms
- Usual—abdominal colic
- Occasional—wrist weakness, joint aches
- Rare—cerebral symptoms

Signs
- Lead line
- Pallor

Laboratory tests
- Hemoglobin
- Stipple count
- Urinary porphyrin test

Certain simple tests are of practical value in confirming a clinical diagnosis of lead poisoning. These tests are the hemoglobin determination, the stipple count, and the urinary porphyrin test.

The urinary porphyrin test is much easier to perform than the stipple count. The porphyrin test has a much clearer "end point," and it is usually positive before the stipple count becomes abnormally elevated. The quantitative advantages of the stipple count, however, would seem to preclude its complete replacement by the porphyrin test.

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The Effect of Altitude on the Pathogenesis Of Tuberculosis

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This paper seeks to make inquiry, by means of a survey of the literature, into the rationale of high altitude in the treatment of tuberculosis, by analysis of the possible effects of altitude on the pathogenesis of tuberculosis. Information will be drawn from three general fields: cellular physiology and biochemistry, animal experimentation, and clinical and laboratory observation and correlation.

The earlier proponents of altitude therapy based their opinions not upon any known effect of altitude on the disease process, or upon any scientific rationale, but rather upon clinical observation. A sampling of literature containing reports of these observations indicates that most such reports covered at most a few cases, and they are seemingly cases with which the author was particularly impressed. Large scale surveys involving large numbers of patients with comparable disease, under comparable conditions, were not made. Many of the proponents of altitude in the treatment of tuberculosis believed that benefit resulted not from altitude directly, but from increase in intensity of the sun's rays, or other forms of therapy with which they were concerned.[1, 2, 3, 4]

In more recent times climatic therapy has largely fallen into disrepute. Articles in medical literature pertaining to these forms of therapy, and more particularly to altitude therapy, are indeed few in number. Interest and effort have been turned to chemotherapy and other methods based upon a more sound rationale.

Lately, however, evidence has come to light which provides a sound rationale for altitude in the treatment of tuberculosis. At least one modern compilation of data on a relatively large and complete scale has indicated that altitude has a definite beneficial effect on tuberculosis.[12]

If any factor is to impede or halt the progress of tuberculosis in the body, it must do so by one of two means, by inhibiting the growth or metabolism of the tubercle bacillus, or by increasing the ability of the cellular defense mechanisms of the body to contain and kill the invading organism.

It has long been known that the tubercle bacillus is sensitive to the oxygen tension of its environment.[5, 6, 7, 9] In 1938 the experiments of Walter Kempner clearly outlined this relationship and put it on a quantitative basis.[6] Kempner's experiments may be graphed to show a remarkably smooth curve indicating the rate of metabolism of tubercle bacilli at varying oxygen tensions. The curve shows that at a certain point reduction in oxygen tension produces a precipitous drop in the rate of metabolism of the bacilli.

At sea level the oxygen tension in the alveolar spaces is about 100 mm. Hg., and Kempner has estimated, at sea level, the oxygen tension inside a tubercle to be approximately 50 mm. Hg. This reduction in oxygen tension is due to the fact that a gradient of tension must exist to achieve transfer of oxygen, since a gas will diffuse only from a region of higher tension to one of lower tension. A series of tension gradients exists between alveolar spaces and the inner layers of a tubercle. If the oxygen tension in the alveolar spaces is reduced, the series

Research paper from the Department of Pathology.

of tension gradients must be maintained, and the decreased oxygen tension transmitted to the end of the line, the inner layers of the tubercle. The body reacts to decreased oxygen tension in the atmospheric air by an increase in the number of circulating red cells and an increase in the concentration of hemoglobin. This permits the circulatory system to maintain the quantity of oxygen transported in the face of the reduced tension or pressure.

The cellular reaction to the tubercle bacillus and the formation of the tubercle are well known. The mononuclear cells which constitute the most prominent part of the body's reaction to the invasion of tubercle bacilli, and its principal means of defense, seem to encounter great difficulty in the phagocytosis of tubercle bacilli and also their destruction after phagocytosis. A mononuclear cell may phagocytose a tubercle bacillus and migrate from the focus of infection only to be overcome by its ingested bacillus and thereby set up a new focus of infection.[8] The mononuclear cells form themselves into a wall about the bacilli.

One might offer many explanations as to how this capsule of mononuclear cells tends to contain the infection and work to the detriment of the bacilli. One explanation might be that this process intends the deprivation of the bacilli of oxygen. If the mononuclear cells cannot readily destroy the bacillus by phagocytosis, this might well be their principal means of combating the tubercle bacillus. The mononuclear cells form themselves many layers thick into the wall of the tubercle. Each cell removes oxygen from its environment so that in successive regions toward the center of the tubercle the oxygen tension must become progressively reduced. Indeed, oxygen at the innermost points must diffuse through the protoplasm of many successive layers of mononuclear cells. This effect is enhanced by the fact that the tubercle is an avascular lesion, in contradistinction to the lesions of most infectious processes.

The zone of lymphocytes which accumulates around the periphery of the epitheloid cells, although taking no part in the phagocytosis of the bacilli, must certainly aid in reducing the oxygen tension on the inside of the tubercle, since they will reduce the amount of oxygen diffusing into the tubercle by metabolizing a part of the oxygen available.

From an evolutional point of view, the oxygen relationship might be considered as a reason for the avascularity of the tubercle. The benefit derived from this avascularity is difficult to explain on any other grounds. By the reduction of oxygen tension alone, however, the tubercle bacilli cannot be destroyed, because it has been shown by in vitro studies that so long as the slightest traces of oxygen are present the tubercle bacilli will remain viable.[7]

If an animal infected with tuberculosis was to ascend to an altitude of 10,000 feet where the oxygen tension of the atmosphere is 110 mm. Hg., and of alveolar air is 62 mm. Hg., a conservative estimate of the oxygen tension within a tubercle would be in the neighborhood of 30 mm. Hg. Interpreted in terms of the results of Kempner's in vitro experiments, this indicates that the metabolic rate of the bacilli would be approximately 65 per cent of the rate at sea level. It is in this range of oxygen tension that the more sharply dropping portion of Kempner's curve is encountered.

Since the general public ascends to the summit of Pike's Peak at about 14,000 feet, even altitudes higher than 10,000 feet (or synthetic altitudes) would seem plausible in therapy with benefit increasing at a steeply rising rate. Altitudes of 6,000 feet have rarely been employed in the treatment of tuberculosis in the past. The reduction of oxygen tension afforded by this and lower altitudes is not sufficient to reach the more sharply dropping portion of Kempner's curve, and hence the reduction in the metabolic rate of the tubercle bacillus at such an altitude is quite small. This may be an explanation of the controversial results of such treatment.

Animal experiments give conflicting evidence. Barach's[9] experiments with rabbits were inconclusive. David,[10] experimenting with rabbits and dogs, found that low oxygen tension had a definite beneficial effect.

It is well known that composition of the white cell components of blood changes with altitude. The percentage of lymphocytes increases, and the percentage of neutrophiles decreases with an increase in altitude. Although there has been a considerable difference of opinion as to whether differential blood counts and total white cell numbers are of value in indicating anything in tuberculosis, a number of investigators, having done extensive work on the question, believe that there is a correlation between a high neutrophile count and failure of the body in containing and recovering from the disease.[11, 12] Many possible sources of error have been scrutinized and evaluated in arriving at this conclusion.

Boissevain and Chapman[12] reported in 1941 the results of an extensive study of blood counts at two sanatoria; one, Mt. McGregor in New York, at an elevation of 1,000 feet, and the other, Cragmoor in Colorado Springs, at an elevation of 6,000 feet. The care in both sanatoria was comparable. These investigators reasoned that if a given patient was moved to a higher altitude, the number of neutrophiles in his blood count would decrease, and if altitude was not an influencing factor his prognosis should remain the same. If the neutrophile count is used as a measure of prognosis, the decrease would

give a false indication of better prognosis. In other words, the prognosis for a given neutrophile count should be better at Mt. McGregor than at Cragmoor.

Extensive blood counting had been done on approximately 200 patients at each sanatorium. The results found by these investigators was opposite to that deduced above. The tabulated results of their investigations may be plotted as a graph which presents strikingly consistent and smooth curves. With a neutrophile count of 7,000-8,000, 71 per cent of the patients at Mt. McGregor subsequently died, whereas only 18 per cent subsequently died at Cragmoor. These investigators present evidence to rule out humidity, sunshine, and other factors as the critical agent in producing the difference in prognosis, leaving altitude as the sole remaining agent. A rigorous statistical analysis was used in interpreting the data.

SUMMARY

The tubercle bacillus is sensitive to a decrease in the oxygen tension of its environment, and it is increasingly inhibited as the oxygen tension is reduced. As long as the slightest trace of oxygen remains, this factor will not effect the death of the bacillus. It is possible that the basic and most important defense process of the body against the bacillus, the formation of the tubercle, is an attempt by the body to reduce the oxygen tension available to the bacillus and thereby inhibit its metabolism and reproduction.

Whether or not the reduction of oxygen tension available to the tubercle bacillus is the most important result of tubercle formation, it must produce some degree of inhibition of the bacillus. Whether or not this aspect of the body's defense mechanism against the tubercle bacillus can be appreciably reinforced by decreasing the oxygen tension in the environment of the entire body has neither been proved nor disproved. Animal experiments have given conflicting and inconclusive evidence.

Clinical observation and correlation by laboratory studies of the highest quality at two large sanatoria have indicated that altitude has a definite beneficial effect in the treatment of tuberculosis. Further animal experimentation and clinical evaluation are needed.

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The basic characteristic of the Welfare State is that it cuts the historic, traditional tie between a man's income and his own effort.

For some services, we all recognize that it is desirable to cut this tie—public libraries, free public education and certain public health services. But there is no substitute for individual responsibility and incentive and the discipline of the market in the overwhelming majority of human and economic relationships.

An English observer put it succinctly this way: "The central objection of the Left to the reduction of food subsidies is that it hits the poor more than the rich. But the purchase of anything absorbs more of the income of the poor man than of the rich. This is the main reason why men strive to avoid poverty. They can avoid poverty only by working harder and producing more; and their efforts tend to make goods cheaper for everybody."

—Emerson P. Schmidt, Director, Economic Research
United States Chamber of Commerce

Sixth Annual Mid-West Cancer Conference

Broadview Hotel, Wichita, Kansas April 1 and 2, 1954

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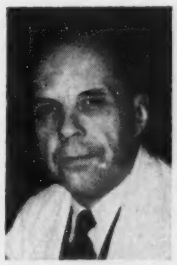
Cornelius P. Rhoads, M.D.
New York, New York

Arthur P. Stout, M.D.
New York, New York

Warren H. Cole, M.D.

Head, Department of Surgery, Illinois College of Medicine; Graduate, Washington University School of Medicine, 1920; Diplomate, American Board of Surgery; Fellow, American College of Surgeons.

Professor of Pathology, Columbia University College of Physicians and Surgeons, New York; Graduate, Columbia University, 1912; Diplomate, American Board of Pathology.



Arthur P. Stout, M.D.

Biographical sketches and pictures of additional speakers will be featured in subsequent issues of the Journal.

Sponsored by
American Cancer Society, Kansas Division
Committee on Control of Cancer, Kansas Medical Society

PRESIDENT'S PAGE

DEAR DOCTOR:

By the time this issue of the JOURNAL reaches you, it will be 1954. For many of you it will mean one more year toward a lifelong ambition of a successful career in the practice of medicine. For many of us it will mean one less year to complete the things that 20 or more years ago we knew in our own minds would be completed, all wrapped up in a neat package, tied with a blue ribbon, and put upon a shelf of our memory as a prize for a job well done. And just the fact that the package isn't finished and wrapped up for many of us, even after 20 or 30 years of practice, is a good reason for all of us to stop and ponder for a few moments.

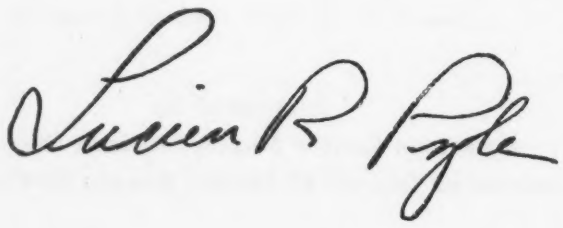
In the lay press and in our conversations with lay people, we are constantly being reminded of the "family doctor." I have, among my personal friends, many who say that they wouldn't know who to call if they suddenly became ill. They bewail the passing of the family doctor in one breath, but they are the same people who, a few years ago, would go out of town to see a diagnostician, or who now go to some distant place to go through a clinic. They may have a diagnosis made and then come home to a friend for an operation, for he is a specialist in surgery and they know him and have confidence in him as a friend and in his ability as a surgeon.

I am sure that the public as a whole is totally appreciative of the age of specialization in medicine and the wonderful advances that have been made in the diagnosis and treatment of disease in the past half century. I am equally sure that not one of the people who criticize the medical profession would be willing to go back to the "family doctor" of 50 years ago, if he had no more to offer them now than he had then. But they would like the companionship and the friendly banter that have been lost to them in the changing of the times and the necessary speeding up of all of our activities.

The old family doctor was a past master in public relations. That is how he got his patients in the first place, and that is how he kept them. In the increasing demands upon the medical profession in the past 10 years, a lot of the niceties of the old "family doctor" have been lost in the shuffle. It is that particular fact that has cast more aspersions on the medical profession than any other one thing. It is a little thing, but it would be easy to correct. It would not take very much of our time. In fact, it would probably lessen the time that we have to spend with patients because they would again develop that abiding faith in us that they had in the "family doctor" of yesteryear.

We hear a lot about public relations. Public relations, as far as the medical profession is concerned, begins in your own office, with you, with your office personnel, and in your social contacts. When this art has again been fully mastered, we will have little need for departments of public relations in our local, state, and national offices.

I wish for each and every one of you and your families, health and happiness, and for you a successful year in the practice of medicine.



EDITORIAL COMMENT

Polio Vaccination Field Trials

On Friday, December 18, in the offices of the Shawnee County Medical Society, a meeting was held that might one day prove to be of historical significance in medicine's attempt to control the incidence of poliomyelitis. On that date, some 30 physicians from various parts of Kansas, representing the Committee on Child Welfare of the Kansas Medical Society, the specialties of internal medicine and orthopedics, as well as pediatrics, officers of at least four among the larger county medical societies of this state, and public health officials, assembled to hear Carl Potthoff, M.D., regional medical director of the National Foundation for Infantile Paralysis.

He suggested that one and possibly two Kansas counties might be included in the national field trial immunizing program to be conducted in 1954, beginning in January. At the conclusion of this meeting, the Committee on Child Welfare met and endorsed the program as explained below. Subsequent endorsement was given by the Council of the Kansas Medical Society. Therefore, if approved by the National Foundation, Kansas will participate in this venture.

Dr. Potthoff spent much time explaining the processes involved in the immunization program and the materials to be used. Jonas Salk, M.D., of the University of Pittsburgh, has developed what appears to be an immunizing agent, and it is his product that will be used. Considerable laboratory testing appears to have demonstrated its successfulness in both monkeys and in human beings. He has at present immunized 700 persons with no untoward effects. He will immunize an additional 5,000 to 10,000 persons before January and before the National Foundation will start its program.

The vaccine is prepared from live virus killed by heat. It appears to be highly protective against Strain I and effective also, if perhaps to a slightly less degree, to Strains II and III. The immunization procedure shall consist of three injections of equal amounts, the first two spaced one week apart and the third consisting of a booster given four weeks after the second.

During the discussion, which continued for two and one-half hours, two principal subjects were discussed. The first of these concerned effectiveness and was quickly disposed of because the answer can be determined only after the completion of this national field trial. It appears effective in the very small number of instances in which the vaccine has been used at the present time. But no conclusions can be reached in that regard until many more persons have been included. The field trial is being performed for the purpose of determining the value of this procedure. It may, therefore, be some years before definite conclusions can be obtained.

The second problem of major interest concerned safety. Here also the evidence is presumed to be satisfactory, but the final determination may be obtained only after many more persons have been immunized. Every known precaution will be taken. Immediate and constant reporting will be required, and at the first indication of an untoward result the project will be cancelled across the nation. There have been no accidents or complications reported to date either in animals or among human beings. Each batch of vaccine will be tested on a minimum of 10 monkeys and in succession through 60 cultures to establish its purity and the fact that the virus has been killed. Every precautionary measure shall be taken and, as stated before, if any accident occurs, the project will be stopped.

The National Foundation will conduct the experiment and will assume responsibility attendant to its procedures. The National Foundation will select the areas in which the experiment is to be conducted. None will be selected on the basis of local requests, but only following the decision of the foundation. However, the test will not be conducted in any locality unless approval has first been obtained from the state medical society and the medical society of the county involved. The National Foundation will supply the vaccine to the state health officer, who will then transmit it to the medical profession within the area where it is to be used. The National Foundation will not supply adjunct materials and will pay no remuneration to physicians or other persons involved.

Moreover, the entire nation-wide test must be completed before June 1. The project will begin in January in the South, where the annual polio season begins early, and will proceed northward. Since the final injection must have been given before June 1, the project must have been started about the middle of May or preferably earlier.

The National Foundation has limited the test to second grade children. Children in the first and third grades will be analyzed for controls. In addition to approval from the medical profession, there must be a written signed request from the parents of each child to be immunized, and certain other records must be kept. Counties selected will probably represent a population of 50,000 or more, and in this state Johnson, Reno, Sedgwick, and Shawnee counties are under consideration. However, this may be altered, but again the decision rests with the National Foundation.

At the conclusion of this meeting, the Committee on Child Welfare further discussed the project and

took action declaring that they consider the procedure to be relatively safe, at least to the degree that they are willing to co-operate with the National Foundation. The action of this committee was then submitted to the Council of the Kansas Medical Society, which body also voiced its approval.

Letters have since been written to each county medical society in this state, requesting local action on this subject regarding the course to be followed should they be invited to participate. This was requested even of the smaller counties which undoubtedly will not be included in the first field trials, but it was felt that each county society should be given an opportunity for expression on this subject so that their opinions would be available if needed. It may be that this may ultimately be discarded. It may be found to have insufficient protective power. It may ultimately be discovered to be dangerous, but it could also be that December 18 may represent a landmark of historic importance in the record of medicine in Kansas.

VA Non-Service Connected Cases

Differences between organized medicine and the Veterans Administration are almost all centered around the disposition of non-service connected cases. The A.M.A. has taken action on this subject and in June, 1953, recommended that medical care of veterans should be limited to:

"1. Veterans with peacetime or wartime service whose disabilities or diseases are service-incurred or aggravated, and

"2. Within the limits of existing facilities, to veterans with wartime service suffering from tuberculosis or psychiatric or neurological diseases of non-service origin, who are unable to defray the expense of necessary hospitalization."

The report further states: "... the provision of medical care and hospitalization in Veterans Administration hospitals for the remaining group of veterans with non-service connected disabilities (should) be discontinued and the responsibility for care of such veterans revert to the individual and the community, where it rightfully belongs."

It appears that non-service connected cases currently represent 85 per cent of all patients discharged from VA hospitals, and that number is growing larger because of the continued liberalization of the VA's interpretation of service connection. When the veteran's condition is once presumed to be service connected, the veteran usually is entitled to the same benefits as one who was wounded in battle. He receives hospitalization, out-patient care, or home town care at the expense of the government and is eligible for financial compensation if his disability is found to be 10 per cent or more.

The veteran whose disability is non-service connected may receive free care only in a veterans' hospital and only if he signs the much publicized pauper's oath. Actually, that is a misnomer because, until recently, he has been required to state merely that he cannot afford to pay for his care. That statement is accepted at its face value and, until now, has not been questioned.

The Congress of the United States, as well as the VA, has repeatedly broadened presumption of service connection, each time adding to the ever growing list of veterans additional persons who may receive medical service at government expense, either in or outside a VA hospital. In 1921, Congress authorized service presumption for all tuberculosis and psychosis cases diagnosed within two years after discharge from service. In 1923, the Congress extended this from two years to three. In 1924, claims for active tuberculosis were considered conclusive and not subject to challenge by the government. A considerable number of other conditions was added to the presumptive list. There followed a brief period in which this was somewhat restricted, but by 1934 these benefits were all reinstated.

By 1948, 23 chronic diseases were specifically listed, and 16 additional tropical diseases were added that had never appeared before. And the VA Administrator was authorized to add such other diseases as he believed necessary. At that time, diagnosis had to be made within a one-year period. In 1950, this was extended to allow a three-year presumptive period for pulmonary tuberculosis. In 1951, multiple sclerosis was extended to two years. In 1953, all types of tuberculosis were extended to three years. In other words, if the disease was diagnosed within three years following the veteran's discharge from service, it was presumed to be service connected.

But all this appears only to be the beginning. Today there are many diseases that shall be presumed service connected if manifest within a year after separation from service. These include malignant tumors, diabetes, epilepsy, nephritis, calculi of the kidney, bladder, or gallbladder, many other conditions, "and such other chronic diseases as the Administrator of Veterans' Affairs may add to the list." Then there are 16 tropical diseases which are presumed service-connected, not only directly but also including any condition or disorder resulting from or originating because of therapy administered in connection with such diseases. The situation goes still further because the law states, "Nothing in this paragraph shall be construed to prevent service connection for any disease or disorder otherwise shown by sound judgment to have been incurred in or aggravated by active service."

The future portends still more. There are a number of bills previously introduced which will await action of the Congress when it reconvenes. Each

would liberalize the presumption period for a particular disease when the disability is 10 per cent or more. One would extend the presumptive period for all chronic and tropical diseases from one to three years. Another extends multiple sclerosis or psychoses to three years. Another extends the period for malignant tumors from one to two years. Another extends the three-year period for tuberculosis to seven years following separation. Still another would make the three-year period for active pulmonary tuberculosis conclusively presumptive so that it could not be contested by the government.

Perhaps there has been some result achieved in the continual opposition to this growing procedure. At least as of November 4, the VA has altered the application form for hospital treatment. Now, in addition to the statement of whether or not the veteran believes he can afford the cost of his care, he is asked to list the value of his property, the current amount of his assets, his average income, and his average expenditures.

On the same date, a letter explaining the use of the new form was sent out by the Administrator of Veterans' Affairs. This circular, Number 11, lists 11 points among which the following might be of interest to physicians. No investigation of any case may be made "except as authorized by Central Office." It states that information on this form is confidential and may not be given to any unauthorized person. Veterans who have been adjudicated to have service connected disabilities will not be required to fill in the addendum. Of more interest, however, are statements 3 and 4 which read as follows:

"3. This addendum may be used in no way whatever to deny hospitalization to a veteran as the law specifically provides that 'the statement under oath of the applicant...shall be accepted as sufficient evidence of inability to defray necessary expenses.'

"4. If a veteran-applicant for hospitalization signs the oath of inability to pay contained in VA Form 10-P-10, that is legal evidence of eligibility for hospitalization and the applicant shall be admitted when a bed is available and the need for hospitalization has been medically determined."

The Facts of Life...

Often times physicians are confronted with a problem of suggesting literature on a particular health problem to the patient. Louis I. Dublin, Ph.D., second vice-president of the Metropolitan Life Insurance Company, has written a condensed question and answer report on many phases of health as well as a wide range of facts relating to man's welfare. The book is entitled "The Facts of Life from Birth to Death."

Each chapter relates to a particular subject such as Population, The Pattern of Marriage, The Sick and Their Care, Cancer, Tuberculosis, Diabetes, other diseases of public interest, etc. The author has expertly chosen his words, and the language throughout the book is understandable to the average lay person.

If one reads an entire chapter, he would find himself well informed on at least the main points of a specific topic, and for those who wish to further their study a selected reference is given at the end of the volume.

Many physicians throughout the state are using the book in their reception rooms and recommending its use to lay persons. The interesting facts and statistics are a result of many years of research by the Metropolitan Life Insurance Company, which regularly makes a substantial contribution to the health of the nation through its services to policyholders, its publications, and its advertisements.

Change in A.M.A. Dues Policy

The House of Delegates of the American Medical Association, during the 1953 midwinter meeting in St. Louis, adopted a significant change in policy regarding the payment of dues. This may affect physicians who have not paid their dues for 1950. It is described in some detail because many Kansas physicians have not elected to become active members of the A.M.A.

This fact is of some interest to the Kansas Medical Society, first because the A.M.A. is the parent body which deserves the support of the members in this state and, second, because delegates to the A.M.A. are apportioned according to the number of members of the A.M.A. rather than on the basis of the number of physicians practicing within the state. Each state is afforded one delegate to the A.M.A. for each 1,000 members of that body.

In 1950 the A.M.A. voted to assess each member annual dues of $25. In that action was included a statement that subsequent dues for any physician who was a member of a state medical society in 1950 would not be accepted until the dues for 1950 had been paid. This has resulted in considerable confusion because many doctors have recently determined to join the A.M.A. but have been reluctant to pay that extra year's dues.

The current action of the House of Delegates temporarily changes this requirement. For a period of six months, an eligible physician may join the A.M.A. upon the payment of $25 without the payment of the dues for the year 1950. This applies only to physicians who join prior to July 1, 1954, and will not be continued after that time. Therefore, it is recommended to those members of the Kansas Medical Society who have previously not joined the A.M.A. and who wish to affiliate with that body,

that they send their dues before July 1. An additional advantage will result from such action because the A.M.A. is planning to publish a new directory and will distinguish the names of members from those who do not belong to the A.M.A.

The procedure for paying these dues is relatively simple and may be accomplished without unnecessary delay according to the following formula. A check for $25 made payable to the American Medical Association should be submitted to the secretary of the county medical society to which the physician belongs. This will be recorded and the check will then be transmitted to the Kansas Medical Society. Here also a record of the payment will be kept and the check will then be forwarded to the A.M.A. In due time a membership card will be received. This card will identify the holder as a member in good standing for the year 1954 and will entitle him to attend the meetings of the A.M.A. Already defined as to location are the annual meeting of the A.M.A. which will be held in San Francisco, June 21-25, 1954, and the midwinter meeting to be held in Miami early in December.

Certain physicians are entitled to membership within the A.M.A. without the necessity of paying dues. These exemptions must have been certified by the county medical society and by the Kansas Medical Society, but do not necessarily coincide with the exemptions of dues that pertain to the county and the state societies. In other words, not all honorary members of the Kansas Medical Society are exempt from the payment of A.M.A. dues. However, none will be exempt except upon certification from the local society. The A.M.A. has established six categories for exemption which are listed by code as follows: Group A, exempted by reason of serving a residency; Group B, on the basis of financial hardship; Group C, those in military service; Group D, disability; Group K, permanent retirement, and Group L, all physicians who are 70 years of age or older.

Again, the A.M.A. will not accept the word of the physician that he is in military service, for example. This must be certified by the county and the state medical societies. It is recommended, however, that the individual physician take the initiative for requesting such exemption and report this request to the secretary of his county society. The same applies to members who reach 70 years of age. Here the exemption becomes automatic, but only after certification. It is not sufficient for the physician to inform the A.M.A. of this fact. It must be accomplished by letters from the county and state societies. Here again, the Kansas societies will not immediately be aware of these facts, and it is urged that the individual physician make his request for exemption if, in his opinion, he might qualify under any of the six categories.

The Kansas Medical Society hopes that each member will unite with the A.M.A. This is not mandatory and has no bearing on the individual's status with either his county or the state society. It is important to everyone that all physicians belong to the A.M.A., and this special notice is hereby given because of the temporary change of policy which may provide an inducement for a considerable number of physicians to join at this time.

Annual Meeting, May 2-6, 1954

Although the complete program for the 1954 annual meeting of the Kansas Medical Society, to be held at Topeka, May 2-6, 1954, cannot be announced at this time, plans are rapidly being completed for the occasion. Committees of the Shawnee County Medical Society, host for the session, are meeting regularly to work out details.

In general, the plan of former sessions will be followed. Scientific programs will begin on Tuesday, May 4, and continue through the morning on Thursday, May 6. The annual banquet will be held, as usual, on Wednesday evening. The first meeting of the House of Delegates will follow a dinner on Tuesday evening, and the second meeting will follow a luncheon Thursday noon.

A chronological listing of events includes two meetings on Sunday, May 2. First is the annual meeting of the Board of Directors of Kansas Physicians' Service (Blue Shield). The second Sunday event is the opening of the two-day meeting of the Kansas Medical Assistants' Society.

There will also be two events on Monday, May 3, in addition to a continuation of the medical assistants' program. One is the annual meeting of the Kansas Academy of General Practice, including both scientific and social programs and concluding with a banquet. Of primary interest to sportsmen is a golf and skeet tournament during the day and a tournament banquet at the Topeka Country Club in the evening.

The Woman's Auxiliary to the Kansas Medical Society will hold its annual meeting on the days of the scientific sessions. A number of specialty societies are also planning sessions during the week.

It is recommended that all who plan to attend make hotel reservations now. Rooms are still available at the two major hotels, and reservations may be made through Mr. Harry Lose, manager of the Jayhawk, and Mr. Earl Shore, manager of the Kansan. Motel accommodations may be secured at the Jayhawk Junior, Meadow Acres, Nod-Away courts, and others.

K.M.S. annual meeting, May 2-6, 1954, Topeka.

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1. Hild, A. M.: Schweiz. med. Wchnschr. 71:557, 1941.
2. New and Nonofficial Remedies, J. B. Lippincott Co., Philadelphia, 1953, p. 200.

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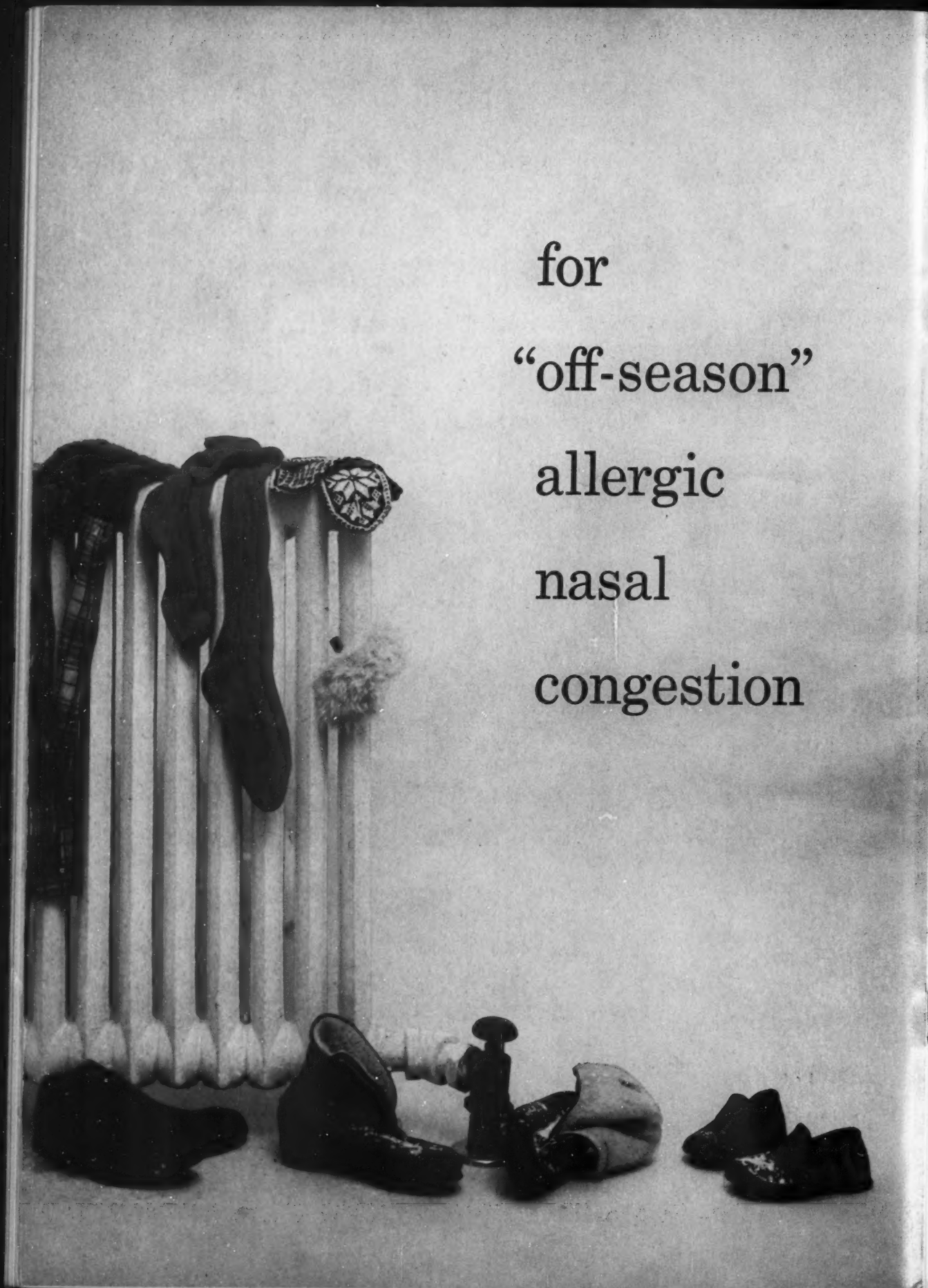
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Adenocarcinoma of the Prostate

Tumor Conference

Edited by Chauncey G. Bly, M.D., and Irwin Joffe, M.D.

Dr. Helwig: Today we have two patients with prostatic cancer; one is without demonstrable metastases or evidence of extension; the other is a relatively young man for cancer of the prostate, and he shows extensive metastases. I would like Dr. Shinkle to give us the essential facts in the history of the first patient.

CASE NO. 53-74

Dr. Shinkle: This 69-year-old white man had had symptoms of prostatism for three years; during the 9 months prior to admission he had noted increased dysuria and frequency. On admission, an enlarged (two-plus), nodular, stony-hard prostate was palpated on rectal examination. The serum levels of acid and alkaline phosphatase were normal. One week after admission, the patient underwent transurethral prostatic resection, and a few days later bilateral orchiectomy was done. He returned home on the 16th hospital day.

Dr. Helwig: I understand that this patient had no significant x-ray findings. May we have the pathologist's discussion?

Dr. Boley: Only a few of the many fragments of prostate tissue submitted show areas of tumor tissue. This relative paucity of tumor tissue in the large sample examined would indicate that either this patient has a rather small carcinoma, or that the region of prostate biopsied contains less tumor than do other regions.

Often as one approaches these areas of carcinoma, he sees a marked tendency for infolding and proliferation of the epithelium in the remaining glands of the prostate (Figure 1). Supporting stroma is completely lacking in these papillary epithelial folds. In some cases, when a pathologist sees only these peculiar proliferations within prostatic acini, so-called "glands within glands," he becomes suspicious, studies further sections and, as in this case, very often turns up one or more foci of unequivocal carcinoma.

CASE NO. 53-75

Dr. Shinkle: The second patient, a 57-year-old white man, was first admitted 9 months ago, after urinary retention had required constant catheter drainage for 5 days at home. He at that time gave a history of "prostatism" of rather short duration, approximately 4 months. The patient walked with a limp, with obvious pain on movement of his left leg. Rectal examination revealed a slightly (one-plus) enlarged smooth, hard prostate. Pertinent laboratory findings included a serum acid phosphatase level of 0.6 millimols (normal 0.1-0.6) and an alkaline phosphatase level of 7.1 millimols (normal 0.7-3.0). After a diagnosis of carcinoma of the prostate had been made on tissue removed by transurethral resection, a bilateral orchiectomy was also recommended, which the patient refused at that time. He was placed on 5 mg. of stilbestrol and dismissed.

Approximately one month later his family physician did perform a bilateral orchiectomy. The patient was seen periodically here in the clinic during the next 7 months. Three days ago he returned to the clinic with increasingly severe pain in his lower back and legs, evidence of extensive metastases. He entered the hospital for further evaluation and for consider-

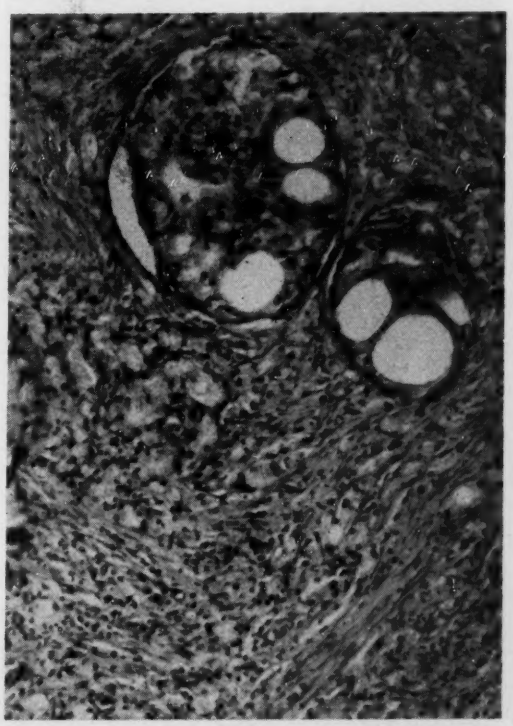

Figure 1. Atypical proliferation of glandular epithelium within two prostatic acini, which are surrounded by sheets of partially differentiated carcinoma cells invading through the prostatic stroma. X 120. Hematoxylin and eosin.

Cancer teaching activities at the University of Kansas Medical Center aided by grants from the National Cancer Institute and the Kansas Division of the American Cancer Society.

Dr. Bly is a scholar in cancer research of the American Cancer Society. Dr. Joffe is a trainee of the National Cancer Institute.

ation of the advisability of therapy with cortisone.

Dr. Litton: Serial roentgenograms of the chest and kidney-ureter-bladder tract during the last 9 months show progression of extensive osteoblastic metastases in the ribs, pelvic bones, and especially in the dorsal spine (Figure 2).

Dr. Helwig: Did examination of the surgical specimens from this case reveal anything different from those of the first case, Dr. Boley, and would you be able to make any predictions from your studies?

Dr. Boley: Microscopic sections from representative fragments of this patient's prostate show moderate numbers of dilated and hyperplastic glands. Much more important, however, are the nests of atypical epithelial cells interposed in great numbers between bands of fibrous and muscular stroma. These epithelial cells vary much more widely in size and shape than do the cells of the typical adenocarcinoma of the prostate as illustrated in the first case. The poorly differentiated or anaplastic tumor cells appear incapable of forming recognizable glands or acini in this second case.

Figure 2. Roentgenogram showing chalky irregular areas of increased density indicating extensive osteoblastic metastases to ribs, vertebrae, and pelvic bones.

The histologic appearance of the tumor cells and glands in carcinoma of the prostate is of little value in estimating duration, present size, or extension of the tumor, or of predicting future potentialities. Even though, in today's cases, there were no demonstrable metastases of the better differentiated carcinoma, and disseminated metastases from the anaplastic one, we still hesitate to prognosticate on the basis of the histologic appearance of cancer of the prostate. Most pathologists today feel that there is little to be gained by struggling to classify carcinomas of the prostate according to the degree of differentiation or maturation of the tumor cells. For example, Foot[1] found no differences in prognosis in patients with diffusely infiltrating anaplastic tumors as opposed to those with well-differentiated carcinoma.

Dr. Helwig: There are several other important areas of discussion in today's topic. Would you lead off, Dr. Valk, by defining prostatism for us?

Dr. Valk: By prostatism we mean (1) increased frequency of urination, (2) decrease in size and force of the urinary stream, and (3) increased effort in voiding. The term comes from the fact that these signs and symptoms are often related to the strategic location of the prostate around the urethra and neck of the bladder. Prostatism occurs in about one quarter of the male population over the age of 55. We feel that it is probably quite normal for a man over the age of 50 to void once at night, but if he gets up more often than this, it is a definite sign of partial obstruction at the bladder neck or of disease of the nerves supplying the bladder. Increased frequency of urination, in the absence of infection or foreign bodies such as stones in the bladder, usually occurs because of failure to empty the bladder at each voiding. With obstruction at the neck of the bladder, the bladder muscle first hypertrophies in an attempt to expel all the urine through the diminished orifice. As long as the increased muscle mass and contractile power can compensate for the partial obstruction, there will be little residual or unexpelled urine. Sooner or later, however, the bladder muscle becomes decompensated, and residual urine will be left in the bladder after voiding.

Three pathological entities commonly cause prostatism: benign prostatic hypertrophy and hyperplasia; adenocarcinoma of the prostate; and vesicle neck contracture.

Approximately 70 per cent of the patients with prostatism have benign prostatic hypertrophy and hyperplasia. The actual cause of this overgrowth of the smooth muscle and the glandular elements in the prostate is not known. However, it is reasonable to believe that variations in the endocrine equilibrium are responsible for the overgrowth, since the prostate in growth, health, or disease is sensitive to endocrine factors.[2] At puberty, the anterior lobe of the pituitary gland produces luteinizing hormone which stimulates the interstitial cells of the testis to produce male sex hormone. The male sex hormone in turn stimulates the prostate to increase in size and to produce secretion. Another pituitary principle, prolactin, apparently helps to mediate these effects.[3] There is very little change in the prostate gland from puberty until about the age of 50 when, most probably because of changes in the pituitary, adrenal glands, and testes, about 25 per cent of the male population develop further overgrowth of the prostate.

It is interesting to note that if a young man is given an excessive amount of anterior pituitary preparation, prostatic hypertrophy will often develop. We have seen pre-pubertal boys with undescended testes who had received excessive amounts of anterior pituitary preparation; all of them had a definite increase in the size of the prostate, which in one of these little boys produced urethral obstruction.

The Sertoli cells of the testes, and the adrenal cortex, probably produce estrogen-like hormones, and as a man grows older there seems to be gradually increased production of these female substances. On the other hand, the male sex hormone, produced by the Leydig cells of the testes, shows an increase in production from the time of puberty to about the 28th year, and thereafter begins a gradual decline. Many people believe that the etiology of hypertrophy of the prostate is somehow bound up with this shift in the androgen-estrogen balance.

Carcinoma of the prostate is the second lesion responsible for prostatism and is the second most common cancer in men over 55 years of age. Approximately 20 per cent of the patients seen in this clinic with prostatism have detectable carcinoma of the prostate. However, only a very small percentage of this group have small isolated nodules of carcinoma which can be satisfactorily treated by removal of the entire prostate and seminal vesicles.

The third lesion responsible for prostatism is contracture of the vesicle neck. This disease also has an obscure etiology. We formerly believed that the scarring at the neck of the bladder was due to previous inflammation and infection in the prostate. Obviously this is not true, since the tissue that we remove in these cases consists mainly of hypertrophied smooth muscle from the neck of the bladder. This lesion is perhaps comparable to the hypertrophy of smooth muscle that is seen in pyloric stenosis in children.

Dr. Helwig: Dr. Valk, would you next discuss digital examination of the prostate through the rectum, and instruct us regarding the clinical grading of the degrees of enlargement?

Dr. Valk: The adult prostate is primarily composed of two lateral lobes and one median lobe. The lateral lobes meet to form a posterior central longitudinal sulcus which is palpable through the anterior rectal wall. A normal prostate is roughly the size of a horse chestnut (3 to 4 cm. in diameter) and weighs from 15 to 28 grams. If the prostate is felt to be slightly larger than the gland just described, it is called "one-plus" enlarged. If the gland is so large that the average examining finger won't go over the top of the prostate and can't reach to the lateral margins, we say the prostate is "four-plus" enlarged. If the finger goes just over the top of the gland and just around the sides, we call it "three-plus" enlargement; and if it's somewhere between one and three, we call it "two-plus." It is difficult to arrive at more exact dimensions by digital palpation through the rectum.

However, the size of the prostate as judged by rectal palpation is not the most important physical characteristic, for the external size and shape may have nothing to do with the symptoms produced. One patient with a small prostate may have severe urethral obstruction due to marked proliferation of periurethral glands only, whereas another individual may have a prostate the size of a grapefruit (9-10 cm.) with practically no urethral obstruction. Furthermore, today's patient with the smaller prostate had many metastases from the carcinoma there.

The most important characteristic of the prostate to be always evaluated clinically is the consistency, which normally is about as soft as the end of one's nose. Any prostate which has focal hard areas or is diffusely hard must be considered to be carcinoma of the prostate until proved otherwise by adequate biopsy. Carcinoma of the prostate, like palpable carcinoma anywhere, ought to be diagnosed earlier than it is. Unfortunately, we see annually fewer than five patients with localized prostatic carcinoma on whom we can confidently attempt a radical operation for removal of the entire prostate and seminal vesicles. If an entire lobe is clinically involved, we usually expect the pathologist to find that cancer has already spread to the seminal vesicles and the regional lymphatics.

Dr. Helwig: Pathologists grade prostatic carcinoma in the following manner.[4] If the cancer is confined entirely to the prostate, it's "Grade One"; when it invades the capsule, it's "Grade Two"; when it reaches the seminal vesicles, it's "Grade Three"; and when it extends further into regional

lymphatics or when it metastasizes to more distant sites, it's "Grade Four." As Dr. Valk mentioned, the only ones that are amenable to surgery are those which we would call "Grade One." These unfortunately constitute less than 5 per cent of the clinical cases of cancer of the prostate.

It is worth mentioning that a year or so ago Hudson[4] examined a group of 59 indigent elderly gentlemen who lived in a Bowery hotel in New York and found that 30 per cent of this group had evidence of cancer of the prostate. This study offers some indication of the remarkable frequency of prostatic cancer in advancing age.

In regard to liaison between the pathologist and the clinician who are attempting to reach a diagnosis on an abnormal prostate, I use what I call the "rule of finger": "If the urologist's finger says that it is carcinoma and the microscope says it's not carcinoma, *it's carcinoma;* if the urologist's finger says it's not carcinoma and the microscope says it is carcinoma, *it's carcinoma.*"

Because so frequently a carcinoma starts in the posterior lobe, it has to extend for a considerable distance before it obstructs the urethra. Moreover, when patients have both benign hyperplasia and carcinoma, as they frequently do, the obstructing element in the prostate may be the benign hyperplastic tissue and not the cancer at all. Therefore, when the urologist relieves such an obstruction with the resectoscope, only a few of the deeper fragments removed may contain carcinomatous tissue, as in today's first case. It is impractical to make serial sections of every fragment received in all cases. Thus, when the pathologist examines what he considers a fair and representative sample of the material, he may miss the areas of cancer entirely. I personally do not hesitate to ask the urologist what his digital examination of the prostate disclosed. If he strongly suspects carcinoma, I section additional material and occasionally will find in one or two fragments a miniature focus of cancer. The urologists understand our problem and inform us of their clinical suspicions.

Now, I would like to have Dr. Valk review for us other aspects of the management of patients with carcinoma of the prostate.

Dr. Valk: In 1941, Dr. Huggins[5] felt that, since the male sex hormone was responsible for the development and maintenance of the prostate, patients with carcinoma of the prostate might benefit by removal of the testes. He was gratified to find that after orchiectomy about 75 to 80 per cent of patients with far advanced carcinoma of the prostate went into remission. At about the same time, it was discovered by Dr. Herbst[6] that orally administered stilbestrol effectively neutralized or inactivated male sex hormones and induced remissions in a similar proportion of patients. Dr. Phil Clark[7] also had been working on endocrinological problems in Dr. Huggins' laboratory and had already discovered that, in this apparent neutralization or inactivation, the female sex hormone was found to be about 10 times as potent as the male sex hormone. People have known for a long time that the female influence was something to be respected, but here was laboratory proof! When we give a male patient 5 mg. of stilbestrol daily, we give him many times more female sex hormone than is necessary to cause changes in the endocrine system that approximate castration.

In 1946, Nesbit and Plumb[8] studied 1,800 patients who had had bilateral orchiectomy and estrogen therapy, and it was discovered that usually within three years and not longer than five years after castration, the patients relapsed. Moreover, the percentage of five-year survivals in the group receiving orchiectomy or stilbestrol therapy or both was no higher than in the untreated group. One explanation that had been previously proposed suggested that the adrenal cortex, behaving like a third testicle, took over some of the function of the testis and produced enough androgenic steroid material to cause reactivation of the cancer. In fact, in 1943-1944, before the days of cortisone, bilateral adrenalectomy had been carried out on three patients, but since at that time adrenal replacement therapy was very difficult, the results could not be accurately appraised.[9]

Finally, with the advent of cortisone, bilateral surgical adrenalectomy has now been performed in about 200 cases of carcinoma of the prostate in this country. Most of the statistics available indicate that this procedure does give palliation in from one-fourth to one-half[10] of the cases but that most of the patients died within 4 to 9 months after adrenalectomy. An even more heroic operation, hypophysectomy, has also been recently tried in a few patients, with too few cases to give unequivocal results.[3]

It has been known for a long time that cortical atrophy and decrease in adrenal cortical function occur following prolonged cortisone therapy. We have recently treated 15 patients with orchiectomy followed by estrogen and cortisone. All of these patients had had far advanced carcinoma of the prostate with many metastases, and had relapsed after first improving on castration therapy. It appears to us at present that the maximum inhibition of far advanced carcinoma of the prostate may be best obtained by a combination of orchiectomy, 5 mg. of stilbestrol a day, and maintenance doses of cortisone.

Dr. Helwig: Have you tried progesterone or other steroids?

Dr. Valk: No, we haven't. Some physicians have tried progesterone and others but felt that the temporary improvement noted was too slight to warrant continued study.[11]

I doubt that the effects of administered estrogens can be satisfactorily explained by a strictly chemical concept of neutralization of male sex hormones. Pos-

sibly the estrogen works upon some such organ as the pituitary, resulting in a depression of the production of gonadotrophic hormones.

Dr. Helwig: I can recall one attempt to explain the effect of administered estrogens, on a more anatomical basis. These investigators[12] noted fibrous sclerosis of the prostate in castrated guinea pigs which had been given estrogens, and they suggest the possibility that this sclerosis might be a direct effect of the estrogen on the prostate.

On the other hand, physiologists have for a number of years speculated as to the ability of the normal body to convert androgen to estrogen or the reverse. Recently a group of investigators[10] at Harvard have found that administered androgens are converted into estrogens in the male, and excreted as such in the urine.

Dr. Bly: Along these lines of thought, I recall that one of the best sources of the original estrone was stallion urine, which may contain 100 times as much estrogenic material as even human pregnancy urine. Strangely enough, the testis of the horse is said to be the richest known tissue source of estrogens,[13] but this does not prove for us that androgens are converted into estrogens in the testis or in any other organ of the body. Certainly such conversion can be done in the chemical laboratory.

Dr. Helwig: Isn't it true that less than one-half of the small localized curable prostatic carcinomas can be diagnosed preoperatively?

Dr. Valk: Yes, I agree. Moreover, these small cancers of the prostate pose difficult problems in management too. A person in his 50's with a small nodule in the prostate is an example of the typical problem-case. We feel that the nodule ought to be removed or biopsied. Sometimes we will use the Turkel needle to obtain a piece of that nodule. However, it is occasionally very difficult, even with one's finger in the rectum, to insert a needle into such a nodule; therefore, a negative result means nothing.

On the other hand, if we are compelled to explore thoroughly the prostatic capsule, we render the patient impotent. Since only about one-half of these nodules are malignant lesions, we must weigh the evidence carefully before proceeding to destroy the nerves of the posterior capsule. Many of these nodules prove to be only fibromas, healed inflammatory areas, prostatic abscesses, or calculi. Consequently, we discuss these matters thoroughly with the patient and explain that sometimes the search for cancer involves the loss of his sexual powers.

Dr. Helwig: I heard Dr. Flocks[14] give his paper at Denver this summer; he had given radioisotopic colloidal gold to 160 patients with carcinoma of the prostate. Can you elaborate on some of his studies for us, Dr. Valk?

Dr. Valk: Dr. Flocks wanted to work out a better method to control far-advanced infiltrating cancer of the prostate. When radioactive gold (Au^{198}) became available, it was discovered rather early that this material, when injected, followed the lymphatic drainage pattern of the area and could often be picked up in the regional lymph nodes and lymphatic channels. Dr. Flocks opens the bladder widely through a suprapubic incision and injects colloidal gold directly into the carcinoma of the prostate. There are many complications to this type of irradiation therapy. However, it often does cause quick regression of the local tumor masses and may even temporarily suppress growth of extensions of the tumor along lymphatics. It offers a method of palliative irradiation of the tumor, with less damage to overlying tissue than x-irradiation would produce. It is possible that one might cure patients of small localized cancers within the prostate using this method, but surgery seems to offer as good or better chances for cures in these cases.[3]

Dr. Helwig: Dr. Tice, would you like to comment on the irradiation treatment of advanced prostatic carcinoma?

Dr. Tice: The only cases I recall in which radiation has been of benefit have been those with bony metastases where pain was a predominant symptom. I am not impressed by a great amount of ultimate benefit to be derived from irradiation in cancer of the prostate, but definite relief of bone pain can be obtained in certain cases with irradiation directly over the metastatic sites.

Dr. Helwig: Has anyone done any bone marrow studies at this institution on early prostatic carcinoma? I recall a study of 60 cases of early carcinoma of the prostate reported a year or so ago.[15] The authors found positive marrow smears in 19 cases, 5 of which showed no evidence of x-ray changes. It was suggested by this group that many of those patients without the x-ray findings or alterations in serum acid or alkaline phosphatase levels might already have metastases that could be discovered by marrow biopsy. The procedure was offered as another diagnostic adjunct to the study of the patient with carcinoma of the prostate before doing radical surgery. Both ilium and sternum were used as biopsy sites with equivalent results. Such studies might be appropriate in our first patient for study today, if he returns to the hospital.

Dr. Tice: I wonder if anyone would comment on the relative infrequency of lung metastases in carcinoma of the prostate, despite metastatic lesions in adjacent ribs.

Dr. Helwig: Pulmonary metastases are relatively uncommon, whereas I suspect that, if we did complete skeletal surveys on all autopsied cases of carcinoma of the prostate, we would find some bone metastases in an extraordinarily high percentage of cases, maybe up to 90 per cent. In Kaufmann's book[16] mere routine skeletal studies disclosed bony

metastases in 50 per cent of cases dying of carcinoma of the prostate. Perhaps the figures would be lower today with better and earlier diagnosis and treatment.

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A.M.A. House of Delegates

The House of Delegates of the American Medical Association, meeting at St. Louis, December 1-4, took important policy actions on social security, voluntary health insurance, medical ethics and unethical practices, medical education, hospital accreditation, military affairs, and a wide variety of subjects affecting both physicians and the public.

Highlight of the opening session was the announcement that Dr. Joseph I. Greenwell of New Haven, Kentucky, had been selected by a special committee of the A.M.A. Board of Trustees as the 1953 "General Practitioner of the Year." The annual medal and citation for community service by a family physician were presented to Dr. Greenwell by Dr. Edward J. McCormick of Toledo, Ohio, president of the American Medical Association, who also addressed the opening session.

The Tuesday program also included addresses by Dr. James R. Reuling of Bayside, New York, speaker of the House of Delegates, and Dr. Chester Keefer of Boston, special assistant to Mrs. Oveta Culp Hobby, United States Secretary of Health, Education and Welfare. Annual reports were presented by Dr. George F. Lull, secretary and general manager of the A.M.A.; Dr. Dwight H. Murray of Napa, California, chairman of the Board of Trustees, and by the standing and special committees of the House of Delegates.

Approving a recommendation by its Reference Committee on Legislation and Public Relations, the House passed a resolution reaffirming its opposition to the compulsory coverage of physicians under the Old Age and Survivors Insurance provisions of the Social Security Act and advocating passage of the Jenkins-Keogh bills now pending in Congress. These bills were described as providing for "the development of a voluntary pension program which is equitable, free from compulsion, and satisfies the retirement needs of physicians."

The reference committee report adopted by the House said: "The purpose of these bills is to eliminate the discrimination and inequities which exist under present tax laws by extending the tax deferment privilege to the country's 10 million self-employed and also to millions of employees who are not covered by pension plans. The purpose of the resolution is to reaffirm our support to the voluntary pension program provided in the Jenkins-Keogh bills and to reaffirm our strong opposition to the extension of compulsory coverage of physicians and other self-employed persons under Title II of the Social Security Act."

The same committee report urged continued action to obtain passage of the Bricker Amendment (S.J. Res. 1) and approved the principle of legislation which would reduce or remove the limitation on the deduction of medical and dental expenses for income tax purposes. It also opposed any further extension of the "Doctor Draft" Law beyond the present expiration date of June 30, 1955.

The report said that "your committee feels strongly that there should be no further extension of the 'Doctor Draft' Law. We feel that the legislation is discriminatory and urge the Committee on Legislation and the Board of Trustees to actively oppose any further extension."

The House acted to accelerate the development of voluntary health insurance by passing a resolution requesting the Council on Medical Service to proceed immediately with a special study of the problems of catastrophic coverage and coverage for retired persons. The Council was asked to present its findings and recommendations to the House not later than the 1954 clinical meeting. The resolution pointed out:

"There are two large groups of citizens for whom improved coverage could be offered under present prepaid medical care plans, namely: (a) those individuals who suffer catastrophic or long-continued and highly expensive illness and whose financial resources are not adequate to meet the cost thereof and (b) those citizens who have retired and are living

on small incomes and who are not eligible under presently existing public or private plans."

The resolution emphasized the medical profession's "responsibility to make every effort to promote such prepaid medical coverage for all citizens whose circumstances make them eligible."

Another resolution on voluntary health insurance, adjudged to be emergency business by the Reference Committee on Insurance and Medical Service and then passed by the House, stated that "the American Medical Association condemns all insurance contracts which classify any medical service as a hospital service." The resolution reaffirmed previous actions of the House defining pathology, radiology, anesthesiology, and physiatry as medical services.

A second emergency resolution, which would have endorsed the principle of federally subsidized scholarships for prospective military personnel in order to encourage the building up of a career-basis medical corps for the armed forces, was referred by the House to the Board of Trustees for study and action.

A resolution introduced by the Iowa State Medical Society, calling for approval of a joint-billing procedure involving services rendered by two or more physicians, was referred to the Judicial Council, at the suggestion of the Reference Committee on Miscellaneous Business, with the recommendation "that the Judicial Council investigate the factors involved in the matter as presented and determine if there are new factors or new facets that would cause it to change the opinion" determined in 1952.

The House approved a revision of one section of the Principles of Medical Ethics of the A.M.A. which clarifies the relationship of physicians to all forms of public information media. The revision had been worked out by the Council on Constitution and By-laws.

In an effort to solve the publicity problems resulting from unethical practices by a small minority of doctors, the House referred to the Board of Trustees a resolution calling for appointment of a special committee with broad professional representation to study all aspects of the problems. The board was asked to study and implement the intent of the resolution and to report its findings to the House at the June, 1954, meeting in San Francisco.

To clarify misunderstandings among physicians regarding the rules and regulations of the Joint Commission on Accreditation of Hospitals, especially as they concern the role of the Department of General Practice in a hospital, the House adopted the following resolution:

"That this House of Delegates of the American Medical Association request the Joint Commission on Accreditation of Hospitals to publish an article, or series of articles, in the *Journal of the American Medical Association* and other official publications circulating among the medical and hospital professions, to acquaint the medical-hospital profession with the regulations, by-laws and their interpretations, and

"That the Commission clarify the methods by which an aggrieved hospital or its staff may appeal a decision with which they are not in agreement."

In the field of medical education the House was "pleased to note" that a fourth grant of $500,000 had been made by the American Medical Association to the American Medical Education Foundation for financial aid to the nation's medical schools. The Foundation reported that its 1953 income now totals $1,174,000 and that the number of contributors now is more than double the total in 1952.

Dr. Louis H. Bauer, of New York, immediate past president of the A.M.A., was elected president of the foundation just prior to the opening of the A.M.A. clinical session. He succeeds the late Dr. Elmer L. Henderson of Louisville, also an A.M.A. past president.

At the opening session of the House, Dr. McCormick in his presidential address made a strong appeal to the nation's physicians for "action that will further the full confidence of the public in our profession."

"Good public opinion cannot be bought," he declared. "It must be earned through exemplary conduct and genuine service in the public interest. Whatever money the A.M.A. and its constituent societies spend for public education and public relations is wasted unless individual physicians take wholehearted interest in assuring the success of these ventures."

Dr. Reuling, emphasizing that much serious work remains to be done, warned that "times are just as troubled as when we had blanket bills before Congress which would have socialized the practice of medicine."

Dr. Keefer told the House that "the voluntary way has been the most successful in the past and there is no reason to believe it will not continue to be in the future." He urged maximum effort, co-operation and leadership on the community level.

Just prior to the clinical meeting the Joseph Goldberger award for outstanding contributions in the field of clinical nutrition was presented to Dr. James Somerville McLester of Birmingham, Alabama, a practicing physician for more than 50 years. The award was presented by the A.M.A. through its Council on Foods and Nutrition.

Final registration at the St. Louis Clinical Session was expected to total approximately 7,500, including about 2,700 physicians.

LAURENCE S. NELSON, M.D.
JOHN M. PORTER, M.D.
Delegates from Kansas

Magazine Approves A.M.A. Stand

An editorial upholding the stand of the A.M.A. on the subject of medical care for veterans with non-service-connected disabilities appeared in the October issue of *America,* a Jesuit magazine. A portion of the editorial is quoted below.

"Dr. Edward J. McCormick, president of the A.M.A., has repeatedly voiced opposition to this government largess. Behind the A.M.A. opposition lies the fear that socialized medicine may get into this country through the back door of the Veterans' Administration. Present veteran population is about 20 million and growing at the rate of one million a year. If free medical service in state-owned hospitals from state-salaried doctors becomes the rule, the government, the doctors claim, will soon control medicine. A.M.A. fears on this point seem well-founded. More than $500 million was spent on veterans' medical care last year, of which only $178 million went for the care of service-connected disabilities. Besides all this there is another big objection to the present law. The General Accounting Office, checking a test sample of 350 patients recently discharged from VA hospitals, found incomes ranging from $4,000 to $50,000. Twenty-five of these veterans had real property and other assets of from $20,000 to $500,000. Yet all had presumably sworn that they were unable to defray expenses. As an editorial in New York Medicine (August 20, 1953) remarks: 'Seldom has lying been so amply rewarded. Seldom has perjury been so widely condoned.' The A.M.A. deserves strong support on this issue."

A pure compound which temporarily lowers blood pressure in animals has been isolated from rhododendron leaves by scientists of the National Heart Institute. Chemically, the compound is pure, but its structure is a mystery. Called andromedotoxin, the substance resembles the veratrum alkaloids in physiological actions, but it is not nitrogen-containing, as alkaloids must be. It has a strong but brief hypotensive effect in low doses.

The drug has never been tested clinically, but it may have therapeutic possibilities. Although it will not be a cure for hypertension, it is felt that it may be found useful in lowering blood pressure in certain cases of hypertension.

Serves on A.M.A. Committee

Dr. John M. Porter, one of the Kansas delegates to American Medical Association meetings, served as chairman of the Committee on Insurance and Medical Service at the A.M.A. session in St. Louis last month. The committee had a number of important services to perform, and Dr. Porter received many commendations on his work with the group.

Dr. Theodore G. Klumpp, president of Winthrop-Stearns, Inc., pharmaceutical manufacturer, has been appointed a member of the Task Force on Medical Services of the Commission on Organization of the Executive Branch of the Government, under the chairmanship of former President Herbert Hoover. The commission, originally authorized by Congress in 1947, was recreated last July.

Objectives are to: provide better medical care for beneficiaries of the federal medical program, create a better foundation for training and medical service in federal agencies, reduce the drain of doctors away from private practice, provide better organization for medical research, and provide a better state of medical preparedness for war.

Eighty per cent of vehicles involved in fatal traffic accidents in 1952 were traveling straight ahead.

95th annual meeting, Kansas Medical Society, Topeka, May 2-6, 1954.

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DEATH NOTICES

Charles Frederick Menninger, M.D.

Dr. C. F. Menninger, 91, founder of the Menninger Foundation, died at his home in Topeka on November 28 after an illness of several weeks. He began practice in Topeka shortly after his graduation from Hahnemann Medical College and Hospital, Chicago, in 1889. His plan to found the Menninger Clinic was realized in 1919 when his son, Dr. Karl Menninger, returned to Topeka after serving a residency in neurology and psychiatry at the Boston Psychopathic Hospital. Another son, Dr. William C. Menninger, joined the group upon completion of his medical studies in 1925. The Menninger Foundation was incorporated in 1941.

Dr. Menninger, who had been an honorary member of the Shawnee County Medical Society since 1940, was an active member for many years and at one time served as president of the group. In addition, he held membership in the American College of Physicians, the American Psychiatric Association, and the Central Neuropsychiatric Association.

ACTIVITIES OF MEMBERS

Dr. Glenn R. Peters, president of the Wyandotte County Medical Society, was guest speaker at a meeting of the Personnel Managers' Club of the Kansas City Chamber of Commerce at the Town House Hotel on November 20. His subject was industrial medicine.

Dr. Roscoe T. Nichols, Hiawatha, was honored by the Masonic Lodge in that city on November 17 in recognition of 50 years of membership.

Physicians of Emporia were guests of St. Mary's Hospital, Emporia, on the occasion of the hospital's 25th anniversary. At a program which followed the dinner celebration, Dr. Thomas P. Butcher presided. Other speakers were Dr. F. A. Eckdall, Dr. Clyde Wilson, and Dr. J. J. Hovorka.

Dr. A. E. Hiebert and Dr. H. W. Brooks, Wichita, were speakers before a dental convention held in Wichita last month. Both spoke on "Cancer of the Mouth."

Dr. H. J. Stacey, who has practiced in Leavenworth for 57 years, announced his retirement recently. He and Mrs. Stacey have moved to Oklahoma City to make their home with a daughter.

Dr. Franklin D. Murphy, chancellor of the University of Kansas, was one of the speakers at a conference of presidents and presidents-elect of state medical auxiliaries in Chicago last month.

Dr. Glen C. Hutchison, Hays, and Dr. Lorin E. Dickelmann, Hutchinson, were speakers at a meeting of the Kansas Medical Technologists' Association at Hays last month. Dr. Hutchison spoke on "Laboratory Analysis in Diabetes," and Dr. Dickelmann discussed "Clinical Aspects of Flame Spectrophotometry."

Dr. W. G. Norman and Dr. Fred Gasser, veteran Cherryvale physicians who have practiced there 45 years and 34 years, respectively, were honored at a community dinner in Cherryvale on November 19. Sponsor of the event was the local Chamber of Commerce. A physician new in Cherryvale, Dr. Lee Kramer, was introduced. Speaker of the evening was Dr. R. G. Carter, Independence, president of the Montgomery County Medical Society.

Dr. D. S. Roccaforte, Hanover physician now stationed with the Air Force at Smoky Hill Air Base, Salina, was recently promoted to the rank of captain.

Dr. F. H. Buckmaster, Elkhart, described emotional illnesses to the Lions Club at Elkhart at a meeting held last month.

Dr. Ward E. Benkleman, who has been practicing in St. Francis during the past two years, has moved to Folson, Montana, and will soon open an office there.

Taking part in a Cancer Nursing Institute held at St. Francis Hospital, Wichita, early in December were the following Wichita physicians: Dr. Christine Thelen, Dr. James H. Holt, Dr. Hoyt C. Blaylock, Dr. Dale W. Peters, Dr. A. E. Hiebert, Dr. Jack W. Graves, Dr. Edward X. Crowley, Dr. John R. Kline, and Dr. W. G. Cauble.

Among the speakers at a meeting on lung cancer held at the sanatorium at Norton on November 22 were Dr. Martin J. FitzPatrick and Dr. Galen M. Tice, University of Kansas Medical Center; Dr. John L. Lattimore, Topeka pathologist, and Dr. George W. Jackson, Topeka, director of state institutions, State Department of Social Welfare.

Dr. Stephen John Kelly, Wichita, has been discharged from the U. S. Air Force and has begun a residency at the Jefferson-Hillman Hospital at Birmingham, Alabama.

Dr. Raymond A. Schwegler, assistant clinical professor of gynecology and obstetrics at the University of Kansas School of Medicine, and Dr. Karl A. Menninger, Topeka, were among the speakers at a psychosomatic institute in Topeka in December. The program was devoted to a study of the relation of emotional factors to the female reproductive system.

An advisory committee of two doctors to represent each of Kansas City's hospitals in a United Hospitals Building Fund Campaign was appointed recently as

follows: Dr. Albert C. Harms and Dr. Leland Speer, Bethany Hospital; Dr. Victor B. Buhler and Dr. H. H. Hesser, Providence Hospital; Dr. D. N. Medearis and Dr. F. J. Strick, St. Margaret's Hospital, and Dr. William A. Love and Dr. C. W. Alexander, Douglass Hospital.

Dr. William L. Valk, Dr. Ferdinand C. Helwig and Dr. Albert T. Steegman, all of Kansas City, took part in the scientific program at the A.M.A. meeting in St. Louis last month. Among the scientific exhibitors were Dr. V. E. Chesky, Dr. W. C. Dreese and Dr. C. A. Hellwig, Halstead, and Dr. E. Grey Dimond, Kansas City.

Topekans who took part in the program for an institute on cancer nursing held at Stormont-Vail Hospital, Topeka, early in December were Dr. Robert H. Riedel, Dr. A. A. Fink, Dr. C. Henry Murphy, Dr. D. B. Foster, Dr. Robert M. Brooker, Dr. Orville R. Clark, Dr. H. L. Hiebert, and Dr. Willis L. Beller.

Dr. Orville R. Clark, Topeka, presented a paper, "Traumatic Chylothorax," at a meeting of the Western Surgical Association held in Chicago early in December.

Dr. C. J. Kurth, Wichita, took part in a panel discussion on "The Mental Health Problem in Industry" at a meeting of the Kansas Association for Mental Health in Wichita early last month.

Dr. George W. Jackson, Topeka, director of state institutions, has been named to a new committee established by the American Psychiatric Association to certify physicians as qualified mental hospital administrators.

Dr. LeRoy A. Calkins, chairman of the Department of Gynecology and Obstetrics at the University of Kansas Medical Center, presented a refresher course at the Minnesota University School of Medicine on December 3 and 4. He also addressed the Minnesota Obstetrical Society on December 5 and the Chicago Gynecological Society on December 6.

Dr. Charles F. Haughey has completed a three months postgraduate course in surgery and dermatology at the Cook County Graduate School of Medicine, Chicago. He plans to spend January and February in New Orleans for additional courses, and has announced that he will return to his practice in Tribune in March.

Dr. Robert L. Newman, of the University of Kansas Medical Center, recently became a fellow of the American College of Surgeons. Early in December he attended a meeting of the American Academy of Gynecology and Obstetrics in Cincinnati and there conducted a round table discussion on "Cancer of the Ovary."

Dr. Warren F. Bernstorf, Winfield, was recently elected president of the Kansas Association of School Boards.

Dr. William H. Crouch, Jr., Topeka, has completed the examinations of the American Board of Pediatrics and is now a diplomate of the board.

Dr. E. C. Beaty, Parsons, who recently returned after a tour of duty with the Navy, has been appointed assistant division surgeon of the Katy Railroad, to serve at the Katy Employes Hospital, Parsons.

Three Wichita physicians, Dr. Ernest W. Crow, Dr. Philip W. Russell, and Dr. LeRoy Williams, recently became diplomates of the American Board of Internal Medicine.

Dr. Chester M. Lessenden, who was recently released from active duty with the Navy, is now practicing dermatology in Topeka in association with Dr. Ernest H. Decker. Dr. Lessenden took his specialty training in New York City and is a diplomate of the American Board of Dermatology.

At the regular staff meeting of the Community Hospital, Beloit, new officers for 1954 were elected as follows: Dr. H. L. Collins, president; Dr. R. E. Bennett, vice-president, and Dr. R. P. Weltmer, secretary-treasurer.

Alumni Group Elects Officers

Officers of the University of Kansas Medical Alumni Association, elected at a meeting held recently in Kansas City, are as follows: president, Dr. Kenneth Cox, Kansas City; first vice-president, Dr. Alfred J. Horejsi, Ellsworth; second vice-president, Dr. Mahlon H. Delp, Kansas City; secretary-treasurer, Dr. Wray Enders, Kansas City.

COUNTY SOCIETIES

A meeting of the Ford County Medical Society was held last month at the Lora-Locke Hotel, Dodge City, with Dr. M. J. Cox, president, presiding. Speaker of the evening was Dr. Paul H. Wedin, Wichita, who discussed "Surgical Conditions of the Chest."

A scientific meeting of the Sedgwick County Society was held at the Lassen Hotel, Wichita, on December 1. Dr. Sloan Wilson, of the University of Kansas School of Medicine, spoke on "Recognition and Treatment of False Anemias and Polycythemias."

In observance of "diabetes week," the McPherson County Society sponsored a case-finding drive in that county. Free check-ups for diabetes were offered by all members of the society during the week.

The monthly meeting of the Bourbon County Society was held in November at the Burke Street Hospital, Fort Scott. Dr. Henry Aldis was in charge of the program.

A meeting of the Johnson County Society was held at the Hotel Olathe on November 24. The program for the evening was a scientific film on the treatment of peptic ulcers.

Dr. B. C. Gradinger, Halstead, presented the scientific portion of the program at a meeting of the Harvey County Society held on December 7. The subject of his paper was "Unusual Tumors of the Colon."

Dr. Francis T. Collins was installed as president of the Shawnee County Society at a meeting held at the Hotel Jayhawk, Topeka, on December 7. The following officers were elected to serve with him during 1954: president-elect, Dr. D. Bernard Foster; vice-president, Dr. H. W. Powers; secretary, Dr. Willis L. Beller; treasurer, Dr. David E. Gray; member of board of directors, Dr. Dwight Lawson; member of board of censors, Dr. John A. Segerson; members of the medical service board, Dr. Floyd C. Beelman and Dr. Newman V. Treger.

Delegates to the Kansas Medical Society meeting will be: Doctors Francis T. Collins, John Crary, W. H. Crouch, R. Dale Dickson, A. A. Fink, G. Bernard Joyce, Howard U. Kennedy, W. O. Martin, and Walter Mau. The following will be alternates: Doctors Horace T. Greene, Richard Greer, John Kennedy, Donald H. Macrae, Donald Pierce, Ralph Preston, L. L. Saylor, Daniel L. Tappen, and D. J. Weber.

The program for the evening consisted of a dinner, at which the doctors' wives were guests, and a humorous program with Mr. Martin Hughes, Chicago, as speaker.

About 130 members of the Southeast Kansas Medical Society and their wives attended a dinner meeting held at the Hotel Besse, Pittsburg, December 9. Dr. W. Clarke Wescoe, dean of the University of Kansas School of Medicine, was speaker of the evening, and Dr. Lucien R. Pyle, Topeka, president of the Kansas Medical Society, spoke briefly.

Dr. J. Warren Manley was elected president of Wyandotte County Society at a meeting held at the Town House Hotel, Kansas City, on December 15. The following officers were chosen to serve with him: Dr. L. E. Leigh, vice-president; Dr. Lee H. Leger, secretary; Dr. Alfred Steinzeig, treasurer; Dr. P. E. Hiebert, to the board of censors, and Dr. William F. Roth, Jr., Dr. S. R. Friesen, Dr. M. R. FitzPatrick, and Dr. C. Omer West to the group of official delegates.

A talk, "Problems Facing the Kansas Medical Society," was given by Dr. Lucien R. Pyle, Topeka. Dr. H. Penfield Jones, Lawrence, councilor for that district of the state, also spoke.

A meeting of the Montgomery County Society was held at the Booth Hotel, Independence, December 16. Guests at the meeting were Dr. Lucien R. Pyle and Mr. Oliver E. Ebel, Topeka, and the wives of members of the county society. Dr. Pyle was the speaker of the evening.

Officers for 1954 were chosen by the Barton County Society at a meeting held last month at the Gateway Country Club, Hoisington. The following were elected: president, Dr. Marvin O. Steffen; vice-president, Dr. L. J. Robison; secretary-treasurer, Dr. Clair H. Cavanaugh; delegate to state meeting, Dr. Lucien R. McGill, and alternate, Dr. Robert C. Polson.

Dr. Adolph R. Mueller was elected president of the Leavenworth County Society at a meeting held at the Hotel Cody, Leavenworth, December 14. Dr.

Kenneth L. Graham was named vice-president, Dr. Tom W. Graham was re-elected secretary-treasurer, and Dr. G. Ralph Combs was chosen to serve on the board of censors with Dr. Melvin Rabe and Dr. Mueller.

Dr. Robert E. Bolinger, of the University of Kansas School of Medicine, presented the scientific program on protein diet therapy. At the business session, the group approved a plan for a change in the councilor district to which it belongs.

Facilitating Nurse Recruitment

A new tool for those engaged in student nurse recruitment efforts is now available, it was announced recently by the Committee on Careers, National League for Nursing. A 68-page booklet in looseleaf form, *Manual for Student Nurse Recruiters,* was printed through a grant from the National Foundation for Infantile Paralysis.

The manual covers a wide scope of subjects and gives case histories of successful student nurse recruitment efforts. To help meet the health needs of the nation, the flow of new students into nursing must augment the 102,019 students currently enrolled in 1,148 professional schools and the additional 6,500 students now enrolled in practical nursing schools.

Included in the book are sections on how to organize a student nurse recruitment committee on local or state level, importance of community representation on the committee, and a definition of duties for each of the volunteer workers. Among the programs listed are speakers' bureaus, liaison work with school counselors, school librarians, women's clubs, church groups, parent-teacher associations, and civic, service, and veterans' organizations. Special emphasis is placed on scholarships for student nurses who might otherwise be unable to enter nursing.

Pictures of Polio Virus

Authenticated pictures of an isolated poliomyelitis virus were shown for the first time in November before a meeting of the Electron Microscope Society of America. The presentation was made by Dr. A. R. Taylor, research virologist at Parke, Davis and Company, Detroit, in a paper entitled "Electron Microscopy of Poliomyelitis Virus."

The pictures, some magnified 200,000 to 300,000 times, showed the poliomyelitis virus to be a small spherical particle about 30 millimicrons in diameter. One millimicron is equal to 1/25,000,000 of an inch. The unique pictures thus confirmed previous estimates that the polio virus is one of the smallest of all viruses.

Dr. Taylor explained that the Parke-Davis work was done in connection with the development of a polio vaccine and "indicates the feasibility of developing concentrated virus preparations necessary for the production of a vaccine." The company reports that a vaccine will not be ready for general use until after further development and extensive clinical tests, which may require a year or more.

Geriatric Research

A program of research focused on a group of diseases primarily characteristic of the aged has been begun by the William S. Merrell Company, Cincinnati. Studies are being conducted on atherosclerosis, heart disease, high blood pressure, degenerative diseases, mental illnesses, and functioning of the brain. Expansion of research in endocrinology is continuing.

Merrell's gerontological laboratory is the fourth major expansion project undertaken since 1946.

Postdoctoral Fellowships Available

The National Research Council has announced that Eli Lilly and Company is offering postdoctoral fellowships in the medical and natural sciences to physicians who are under 36 years of age. Applicants must agree to devote themselves to research in anatomy, biochemistry, biophysics, histology, immunology, microbiology, pathology, pharmacology, physiology, and related subjects.

Application blanks may be obtained from the Fellowship Office, National Research Council, 2101 Constitution Avenue, N.W., Washington 25, D. C.

Fellowships in Industrial Medicine

Eight fellowships in industrial medicine will be offered by the U. S. Atomic Energy Commission for the 1954-1955 academic year. The fellowship program, begun four years ago, is designed to provide advance training and experience for physicians in the field of industrial medicine.

The program is administered for the AEC by the Atomic Energy Project of the School of Medicine and Dentistry, University of Rochester, Rochester, New York. Applications should be submitted by January 1, 1954, to Dr. Henry A. Blair, AEC Fellowships in Industrial Medicine, at the Rochester address.

Annual meeting, Kansas Medical Society, May 2-6, 1954, Topeka.

THE MONTH IN WASHINGTON

Editor's Note. The following summary of Washington news was prepared by the Washington office of the A.M.A. for distribution to state and regional medical journals.

The second session of the 83rd Congress is getting down to its task under conditions that could mean passage of considerable legislation of importance to medicine. Holding over from last session, or certain to be introduced this year, are bills touching on virtually every phase of medicine where the federal government could become involved. New laws are being proposed on veterans' care, social security, national health plans, care of military dependents, medical scholarships for military personnel, and many other subjects.

What will be done with this mass of legislation depends on an administration whose control over Congress is tenuous and a Congress looking forward to the fall, when all members of the House and one-third of the Senate must be elected or reelected. As is the case every two years, most lawmakers will be listening closely to what's being said back home.

Awaiting congressional action is the administration's plan for extending the social security system to bring more than 10,000,000 additional persons, including physicians, under Old Age and Survivors Insurance (OASI). This legislation is known to have less support in the House Ways and Means Committee, where it is being handled, than it has in the Executive Branch.

American Medical Association, supported by dentists, lawyers, farmers, and many other groups of self-employed, has consistently opposed inclusion under OASI. The question now is whether this opposition will be articulate enough to convince Congress.

In place of social security for physicians, the A.M.A. for several years has actively promoted legislation identified first as Reed-Keogh, then as Jenkins-Keogh, named for the sponsoring congressmen. This would allow physicians and other self-employed to defer income tax payments on a portion of their income, placed in restricted pension funds, obtainable in the form of benefits only in case of disability or at the specified retirement age. In this effort the physicians again are joined by a large group of associations representing the self-employed.

Other possible amendments to the social security law involve total and permanent disability payments and waiver of OASI premiums for the disabled, so their final pensions won't be reduced because of periods when they had little or no income. In each of these, medical determinations would be required. In the past, these bills have threatened an expansion of the federal medical program, have laid out an unreasonable role for the physician, or have called for compulsory rehabilitation. While not opposed to the objectives, A.M.A. has urged that both the patient and the physician be protected. In place of waiver of premium, the A.M.A. proposes that pension rates be based on the 10 best earning years, thus obviating the need for medical determinations.

As in other sessions, Congress this year probably will be asked to pass legislation providing free hospitalization under OASI for all persons past 65 covered by OASI, and for other beneficiaries of the program. In other years Congress has not taken this idea seriously.

The veterans program is certain to provoke action. Last November, Veterans Administration amended its forms to require more financial information from veterans applying for hospitalization of non-service connected disabilities, who must state that they cannot afford private care. Congress may want to further clarify the government's obligation to veterans. It is expected also that special effort will be made to expand medical benefits for veterans by such methods as increasing the periods in which certain diseases may be presumed to be of service origin.

The A.M.A.'s position on the care of non-service connected cases is well known. It consists of three points. First, the best possible care by VA for actual service-connected cases. Second—until local and state facilities are adequate—VA care for long-term tuberculosis and neurological cases when the veteran himself can't pay. Third, all other non-service connected cases to be the responsibility of the veteran himself, his family, or his community.

The Defense Department has served notice that this session it will press hard for implementation of the Moulton Commission's recommendations for broadening the medical care program for military dependents. The Commission favored caring for as many dependents as possible at military installations, with the others receiving private care and the federal government paying all but a token of the cost. At its December meeting, the A.M.A.'s House of Delegates proposed that in this country the military provide medical care for dependents only where private facilities are not adequate.

Also up for decision this year is a Defense Department's proposal that the federal government furnish medical, dental and nursing scholarships, with the recipients obligated for government service at the rate of one year for every year of the scholarship.

There is a strong possibility of pressure to enact a program under which the federal government would

in one way or another subsidize private health insurance plans. The idea is known to interest Rep. Charles Wolverton (R., N.J.), chairman of the House Interstate and Foreign Commerce Committee, which last fall conducted a series of hearings on health matters. Senators Ives (R., N.Y.) and Flanders (R., Vt.) are offering a bill along the same lines in the Senate.

The controversial Bricker resolution holds over from the last session, and may receive early consideration in the Senate. Senator Bricker believes that Congress should have some check on the President's treaty-making powers. The American Medical Association repeatedly has indorsed the Bricker resolution as a safeguard against the introduction into this country by treaty of government-controlled medical plans without Congress itself having a chance to pass on them.

Awaited with interest in Washington are the findings of two Commissions appointed last year to look into the relationships between the federal government on the one hand and state and local governments on the other, and to investigate operations of the executive branch. The former is headed by Clarence Manion and the latter by former President Hoover. The Hoover Commission has until next year to make its report. The Manion Commission was instructed to have a report ready by March, but it may ask for more time.

ANNOUNCEMENTS

The Ninth National Conference on Rural Health, sponsored by the A.M.A.'s Council on Rural Health, will be held in the Baker Hotel, Dallas, March 4-6. The conference will stress the importance of individual participation in community affairs, and the theme will be "Let's Put More 'U' in CommUNITY."

The formal sessions will begin at 2:00 p.m. on March 4, preceded by a pre-conference meeting "for doctors only." That session will be concerned with two items, physician participation at the grass roots and health insurance matters.

The National Multiple Sclerosis Society and the National Institute of Neurological Diseases and Blindness are distributing a new pamphlet designed for consumption primarily by the victims of multiple sclerosis. It is a realistic presentation of the medical status of the disorder and the facilities available for its amelioration.

Physicians who see patients who will find the booklet helpful are asked to advise such patients that the booklet is available from the Public Inquiries Branch, Public Health Service, Department of Health, Education, and Welfare, Washington 25, D. C.

The Third Interim Congress of the Pan American Association of Ophthalmology will be held in Sao Paulo, Brazil, June 17-21, 1954. Meeting concurrently in Sao Paulo will be the Eighth Brazilian Congress of Ophthalmology and the International Congress of Oto-Neuro-Ophthalmology. Translations of all proceedings will be offered in three languages, including English. Attendance is not restricted to members. The registration fee is $10.

The Third International Congress of Internal Medicine will be held in Stockholm, September 15-18, 1954, under the auspices of the International Society of Internal Medicine in which 40 nations are represented.

The 18th annual midwinter postgraduate clinics sponsored by the Colorado State Medical Society for the Rocky Mountain region will be held at Denver, February 16-19, 1954, with headquarters at the Shirley-Savoy Hotel. The program, designed especially to interest general practitioners, will consist of three mornings of "dry" clinics, three noon round-table luncheon discussions, and three afternoons of illustrated lectures by guest speakers. A dinner dance will be held on the evening of February 18.

Registration is open to all doctors of medicine and their guests. A fee of $5.00 will be charged, but students, interns, full-time residents, doctors' families and other non-physicians are exempt.

The International Academy of Proctology announces its annual cash prize and certificate of merit award contest for 1953-1954. Awards will be given for essays on proctology and allied subjects, not exceeding 5,000 words in length. Entries must be received no later than February 1, 1954. The address is International Academy of Proctology, 43-55 Kissena Boulevard, Flushing, New York.

A sectional meeting of the American College of Surgeons will be held March 1-4, 1954, at Omaha, Nebraska, with headquarters at the Hotel Fontenelle. Each morning there will be clinics at Omaha hospitals, and each afternoon will be devoted to scientific programs. The faculties of the medical schools at the University of Nebraska and Creighton University are co-operating in the presentations.

Because of the popularity of this type of program, an invitation to attend is extended to all surgeons, without regard for fellowship in the College. Hotel reservations should be made immediately.

BLUE SHIELD

New Enrollment Experiment

A new Blue Cross-Blue Shield community enrollment program for 1954 has been approved by the Boards of Trustees, and as a result annual enrollment dates will be established in every county.

In other words, Kansans will now know ahead of time when Blue Cross-Blue Shield will be available in their area. These dates will be the same each year in every county.

Employee group enrollment will continue to be available at any time. Groups may be formed in companies having five or more employees by meeting regular quota requirements.

The new method of community enrollment is made possible by the introduction of a new application form which contains a fairly detailed and comprehensive statement of health. The gist of the health statement is as follows:

1. Has anyone to be covered by your application for membership had or does he now have: cancer, diabetes, asthma, epilepsy, ulcers, high blood pressure, kidney diseases, rheumatic fever, thyroid trouble, physical defects (congenital or otherwise), heart trouble, arthritis, tuberculosis, paralysis? If "yes," please give details (including date of last consultation or treatment, attending physician and address, name of hospital if hospitalized).

2. Has anyone to be covered by your application for membership been hospitalized within the last three years for any condition other than those listed above? If "yes," please answer fully (full name of person, relationship to applicant, condition or ailment, name and address of hospital, name and address of doctor).

3. Give names of regular family physicians.

4. Do you consider all persons to be covered by this application to be in good health and free of disease? If "no," please explain.

5. I authorize any physician or hospital to furnish Kansas Blue Cross-Blue Shield information which may be pertinent to the medical history of all persons to be covered by this application.

6. I have read paragraphs one through three, and to the best of my knowledge, the above information supplied by me is correct and complete.

I further understand that acceptance of this application will be based upon the information contained in it and that any misrepresentation or failure to include information concerning the persons for whom application is made will give Kansas Blue Cross-Blue Shield the right to deny benefits for any conditions misrepresented or not reported on this application.

The purpose of this health statement is to improve the selection of risk among new members. In the past, Blue Cross-Blue Shield has been wide open to those who were in poor health and were perhaps the first to apply for membership in every community enrollment. The health statement will tend to prevent this anti-selection and will put emphasis on people joining before they become poor health risks.

If an applicant checks that he has one of the conditions listed on the application, one of two decisions will be made by the Underwriting Section of Blue Cross-Blue Shield:

1. Whether to accept the membership at all.

2. Whether to accept the membership by attaching a rider to the contract excluding coverage for any medical or hospital care for the condition checked.

It is believed that this manner of handling applications will cause the least misunderstanding and poor public relations later on. The member will definitely know that his contract is not good for the condition specified in the rider. He will know this before he has paid his dues. Other members of the family are not affected by the rider.

In spite of every effort which will be made to have a clear understanding with new members, the utmost co-operation by physicians will be necessary to inform people of the spirit and intent in this new program. Some people are going to resent the health statement. Perhaps some doctors will not like to give information which may prevent their patients from receiving benefits. But the health statement is necessary if a more open enrollment plan is to be followed. The only safe alternative to a strict health statement is group enrollment on a quota basis. Of course, this would be virtually impossible in the non-group category.

This new enrollment program is both more liberal and more strict. It is more liberal in the sense that it will open up enrollment to every county in the state (except Wyandotte and Johnson) at least once a year. It is more strict in that the health statement is being required which will tend to screen out poor health risks.

The understanding of the medical profession is earnestly sought so that the program may be successful.

Attention is called to an error in last month's Blue Shield column. Income limits for service benefits have not been changed. They are $2,000 for the single member and $3,000 for the family member.

Twenty-seven million people are now enrolled in Blue Shield medical care plans, according to an announcement by the Blue Shield Commission in Chicago.

Radioactive Iodine in Hyperthyroidism

Lowell Rhodes, M.D.

Wichita, Kansas

The etiology of hyperthyroidism is still unknown, and, therefore, it is understandable why its treatment remains empirical. The present methods of treatment consist of interruption of the production of thyroxin by antithyroid drugs, destruction or removal of all or part of the thyriod gland by surgery, or radiation of the gland. We are awaiting some drug or some method that will attack the disease at its source and thus make the present methods of treatment obsolete.

It might be said that the development of radioactive iodine, for use in the treatment of diseases of the thyroid gland, really began in 1915-1916, when David Marine[17] definitely established the affinity of the thyroid gland for iodine. The first studies[21] with radioactive iodine itself were done in 1934 by Fermi,[36] who developed it by exposing iodine to tubes containing radium and beryllium powder for 30 minutes. Radioactive iodine, itself, did not become readily available until 1936, following the development of the cyclotron. Fourteen radioactive isotopes have been identified, of which four have been used in biological investigation:[21] I^{126} with a half-life of 13 days; I^{128} with a half-life of 25 minutes; I^{130} with a half-life of 12.6 hours; and I^{131} with a half-life of 8 days.

Among the first institutions to start investigations in 1937 were the Massachusetts Institute of Technology and the Massachusetts General Hospital. They were soon followed by the University of California and others. These studies have done much to solve the problems of the metabolism of iodine and the synthesis of thyroxine as well as the mode of action of some antithyroid drugs. Among the early reports of the use of radioactive iodine in hyperthyroidism were those of Hamilton and Lawrence,[10] and Hertz and Roberts,[13] using a mixture of I^{130} and I^{131}. Hamilton and Lawrence used large doses of I^{131} and reported the complete destruction of the thyroid without damage to other organs in rabbits and dogs. Later they gave smaller doses to human beings, with a resultant drop in the basal metabolic rate.

Miller et al.[18] have stated certain conditions which must be fulfilled in order for any radioisotope to be usable for the therapy of disease. First, the disease process must be capable of being treated successfully by a radioactive substance. The isotope being used must have a physical half-life long enough to be useful, but not so long as to make its use dangerous. It must be concentrated in the tissue being treated, but not in any tissue in dangerous quantities. It must remain in the tissues long enough to permit effective radiation. Its main radiation must be from alpha or beta particles, so that its radiation effects are largely limited to the tissue treated. Small portions of gamma rays should be present, so its activity may be measured outside of the body.

Because of its convenient half-life, and the relatively pure form in which it can now be obtained from the uranium chain reacting pile, I^{131} with a negligible contamination of I^{133} is now almost universally used in both investigation and therapy.[31] With quantities of radioiodine, as discussed later, neither objective nor subjective evidence of injury to the kidneys, liver, salivary glands, or stomach has been reported,[18] and it is to be considered safe in the light of our present knowledge.[4, 32] Even overdoses, up to 100 times the therapeutic dose, do not seem to damage other organs.[6] The possibility of late radiation damage leading to malignant change cannot be ignored; however, a concensus of opinion based on experience with x-ray treatment of toxic goiter indicates that late malignant complications with radioiodine therapy are probably unlikely.[32]

Radioactive iodine is given orally. Absorption begins immediately, is about 80 per cent complete in one hour, and is usually complete in two hours.[15] If food is present in the stomach, absorption is delayed. A curve of radioactivity in the blood (radioactive protein bound iodine) has been plotted by Keating,[15] and is seen to have three components: (a) A rapid rise, followed by a brief, rapid fall. This phase represents absorption and the establishment of equilibrium between the blood and other body fluids; (b) A more prolonged fall, the rate depending upon the state of the thyroid and upon renal function. In this phase, if the function of the kidneys is altered, then the renal collection rate is lowered and the fall in the blood is more prolonged;[16] and (c) A flattened out phase, with an occasional rise in hyperthyroid patients. The peak in this curve is reached in myxedematous and normal subjects in two hours, while the peak in those with hyperthyroidism may not be reached for eight hours.[15]

Following absorption, the concentration of the isotope is 10,000 times greater in the thyroid tissue than in any other tissue, according to Crile.[6] From 85 to 90 per cent of the total amount given in a

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This is one of 11 senior theses selected for publication by the Editorial Board from a group of 15 judged the best by the faculty at the University of Kansas School of Medicine.

Thesis written while the author was a senior student. Dr. Rhodes is now interning at Wesley Hospital, Wichita, Kansas.

tracer dose can be accounted for in the thyroid and excretion in the urine,[16, 32] the urine being the principal route for its disposal in euthyroid and hypothyroid subjects.[16] Sixty-five per cent is excreted in the urine in two days. Almost complete excretion occurs in hypothyroid patients in 4 to 5 days, while normal or hyperthyroid individuals may require several days to weeks for complete excretion.[16] If the urine is collected and tested, a reciprocal relationship is noted between the rate of uptake and the amount excreted.[33] Despite the large amounts of radioiodine that pass through the kidneys, no adverse effects have been noted upon them.[18]

Small amounts are also lost in the feces (1 to 2 per cent) and in sweat. About 10 to 15 per cent is distributed to the various organs and tissues of the body other than the thyroid.[16] By the end of one month, most of the radioactivity is gone, and by two or three months, practically none remains.[6]

The metabolism of radioactive iodine can be dramatically influenced by a variety of things. Thyroid extract given before administration causes a depression in the rate of uptake. Werner[33] noted that as little as .12 grams permitted an uptake of only 4 per cent. He and others[22] have shown that iodine lowers the uptake. The normal values of uptake for adults are not necessarily those found in children,[24] who may show a low uptake but are not clinically suspected of being hypothyroid. And then in normal aging, there is a decrease in the basal metabolic rate which may be correlated with atrophy of the acini, decrease in colloid and iodine content, and a decrease in the number of dividing cells.[22] Sex plays a part, too. The rate of uptake of young males and females may be about the same, but the mean gradient of the females is higher than the corresponding mean of the males in both the young adult and the older age groups.[22]

The rate of uptake also varies in certain hormone disturbances. Werner[33] reports a lowered uptake in panhypopituitarism and in adrenal cortical hyperfunctioning, while Reiss[26] reports a decrease in adrenal cortical insufficiency. The uptake is reported by Reiss[26] to be increased when given simultaneously with .3 mg of epinephrine. Sofer, Gabrilone, and Jailer report a decreased uptake of I^{131} when given with epinephrine in intact rats, but the uptake is markedly increased if I^{131} is given simultaneously with epinephrine in adrenalectomized rats.[30]

Dyes containing iodine used in x-ray studies will influence the uptake for varying periods of time. Those used in gallbladder visualization and bronchography will affect uptake for several months, while the effects of those used in intravenous pyelograms last several weeks.[31] Most of these factors which cause variations can be elicited on questioning or examination and should not be of great moment to the clinician when they are known.

In recapitulation, the use of radioactive iodine in hyperthyroidism is justified by its collection by the thyroid gland shortly after administration and the rapid excretion by the kidney of that not taken up by the gland. The thyroid gland can be destroyed by I^{131} in doses that show no damaging effect upon any other organ or tissue in the body. And lastly, the clinician must be aware of the conditions which will produce a variation in iodine metabolism.

Having determined that we have an agent for use in thyroid disease, the next question is, how shall we use it in thyroid evaluation? There are three methods of study open to us. First, we may measure the percentage uptake of radioactive iodine by the thyroid during a given period of time under standardized conditions. This will be discussed later. Second, we may measure the urinary excretion of the isotopes in a given period of time under controlled conditions. This method will not be considered here, due to inaccuracies which are attributed to improper collection of the specimen, failure to collect a complete specimen, and the more technical task of determining the radioactivity.[16, 33] The third method is that of radioautographic prints of tissue. This method, though of little use to the clinician in his office practice, will be discussed because of its value in helping us to understand some of the problems of thyroid disease.

Dobyns and Lennon[7] and Dobyns, Skane, and Maloof[8] have noted a difference in the state of functioning of thyroid adenomas with radioautographs of these tumors. Their method consists of giving patients I^{131} 24 to 72 hours before surgery, removing nodules, making blocks, cutting and mounting tissue, and then exposing the tissue to film for varying lengths of time to obtain prints. They make the assumption that the degree of affinity for radioactive iodine represents the degree of function of the thyroid tissue. Then, by staining and studying the slides, and comparing the slides with the radioautographs, they have noted several types of adenomas.

There are adenomas which have cellular hypertrophy and hyperplasia and which hyperfunction, and there are also adenomas which have cellular hypertrophy and hyperplasia and which scarcely function at all. The group with little function shows an increased variability in cell height and appears to shade off into carcinoma.[7] Dobyns, Skane, and Maloof[8] constructed a Geiger counter suitable to count small areas of a gland, permitting them to determine the activity of a nodule. Then it may be concluded[6] that if a single nodule is found to have distinctly less function than the extra-nodular tissue, this will exclude the hyperfunctioning group and increase the suspicion of malignancy. Without such a special Geiger counter, the clinician cannot tell the activity of the nodule and will have to rely completely on his clinical judgment in therapy.

The usual method of determining the percentage uptake of radioactive iodine by the thyroid gland is initially a determination of the activity of 40-100 microcuries of I^{131} with a Geiger counter. The agent is then given orally to the patient in water, in a paper cup. The cup is rinsed with water, and the patient drinks the rinse water. The patient returns in 24 hours, at which time the activity is determined over the thyroid gland, again by means of a Geiger counter,[11] and the percentage uptake is computed. Werner, Quimby, and Schmidt[32] found the normal uptake in 24 hours to lie between 10 and 40 to 50 per cent, with a mode between 15 and 30 per cent. In nearly all cases of hyperthyroidism, more than 40 per cent of the administered dose was found within the thyroid at the end of 24 hours; in subjects with myxedema, the corresponding figure was usually less than 10 per cent.[33] If the urine excretion should be calculated, it is found that euthyroid subjects excrete from 52 to 84 per cent of the administered dose with an average of 66 per cent in the urine.[15] For thyrotoxic subjects, the range of excretion was 6 to 32 per cent with an average of 18 per cent. Hypothyroid subjects excrete 35 per cent of the dose.[11] The results should then be correlated with other clinical data.[33]

Having decided that a patient has hyperthyroidism, careful selection of patients to be treated with radioactive iodine should be made, in order to secure satisfactory results. Since therapeutic activity depends upon beta radiation, which penetrates tissue for little more than two millimeters,[4] the major field of usefulness[1, 4, 20] is in the diffusely hyperplastic goiters which cannot be more readily and satisfactorily treated in some other way. The selected patients should be those: who fail to respond to apparently adequate dosage of an antithyroid compound, or who have repeated relapses after such treatment; who are sensitive to antithyroid compounds; who would be a surgical hazard due to age, heart disease, bleeding dyscrasias, and such; who have a recurrence of toxicity after repeated surgical procedures; or who have had damage to a vocal cord. Because the possibility of causing a malignancy due to radiation cannot be completely discounted, the majority of investigators prefer to use I^{131} only in patients of the older age group, although malignancies due to radiation are usually associated with chronic, low intensity exposure, and not brief radiation.[2] It is generally conceded that surgery is the treatment of choice in single adenomas where carcinoma may be present;[20] however, I^{131} may be an acceptable type of treatment in patients with nodular goiters in the older age groups who are poor surgical risks, or in those who definitely oppose operation.[20]

Contraindications to I^{131} treatment are few. Since the fetal thyroid is capable of absorbing and concentrating iodine as early as the 14th week, it seems unwise to give an isotope with a half-life of 8 days to a woman who is beyond the 10th week of pregnancy.[3, 5] It has been felt that renal disease represents a condition in which therapeutic doses of radioiodine might do considerable harm. However, Williams et al.[35] have reported no increase in radioiodine in the kidneys of patients with severe glomerulonephritis.

When it is decided that a patient is to be treated by I^{131}, his uptake is determined and all iodine and thiouracil-like therapy is withheld for two[31] to four days.[4, 20] The therapeutic dose is then given in a manner similar to the tracer dose as described above. The amount to be given depends upon:[1] the fraction concentrated in the thyroid; the weight of the thyroid gland; the disintegration scheme of the isotope; the time necessary for decay of radioactivity to zero, and the rate of elimination from the gland.

Obviously the greatest chance for error lies in the estimation of the size of the gland. Shipley and his associates[29] estimate the size of the gland by assuming the normal gland to weigh 20 grams; the lower limit of an enlarged gland is about 35 grams, and the largest gland weighs about 100 grams. A satisfactory dose for therapy probably lies between 100 and 150 microcuries per estimated gram of tissue in diffuse goiters,[4, 28] although all factors must be taken into account in any given case. For nodular goiters the dose is about 240 microcuries per gram of tissue.[28] There are cases reported where as much as 500 microcuries per estimated gram of tissue have been given without permanent myxedema.[14] Some workers determine the dose by estimating the amount of radiation necessary, and then converting this to microcuries, but since they do not agree on the amount of radiation necessary, estimation of the size of the gland is satisfactory.[23, 28]

Clinical improvement usually appears about the middle of the second week, but remission is not established until the end of the second or third month.[19, 29, 32] During this time thiouracil treatment may be necessary. It may be started in from 5 to 7 days after treatment.[31] Actual shrinkage of the gland may not appear for three months.[29, 32] At the present time it appears possible to obtain therapeutic success in 75 to 90 per cent or more of selected patients with hyperthyroidism.[2, 31]

If after two months there is no improvement or only slight improvement, further treatment may be necessary.[19] If the basal metabolic rate is unchanged, the first dose should be doubled. If it is one-half its original amount, give a dose of I^{131} equal to the original dose, and if it is 2/3 of the way to normal, give about one half of the original dose.[6]

If the response in nodular goiters is not what was expected, it may be because:[25, 27] the I^{131} gets lost

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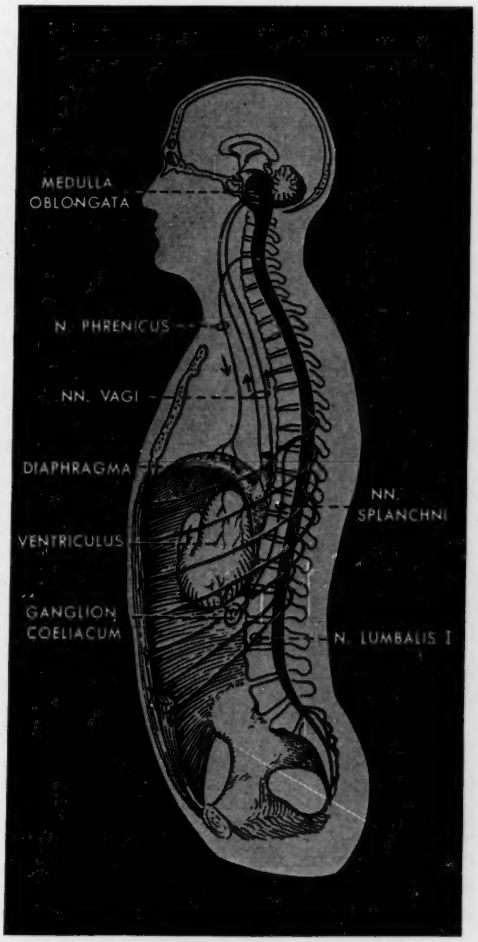

THE VOMITING REFLEX: *Vagus→nodose ganglion→solitary tract→spinal cord→cervical, thoracic and lumbar nerves to diaphragm, cardiac sphincter, stomach, abdominal and pelvic musculature. (After Krieg, W. J. S.: Functional Neuroanatomy, ed. 2, New York, The Blakiston Company, Inc., 1953, p. 104.)*

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in the colloid of a huge nodular goiter, and, being relatively far away from cells, fails to irradiate them effectively; the nodules are in different states of activity, and, as the hyperactive nodules regress, other nodules become hyperactive; nodular goiter is so totally different from the diffuse goiter of Grave's disease that the uptake of I^{131} is totally different, and the response to treatment is different.

Angina pectoris has either disappeared or been greatly ameliorated when hyperthyroidism was controlled. Persistent auricular fibrillation following therapy indicated either continuing hyperthyroidism or severe underlying organic heart disease, necessitating other means of heart control.[25]

A number of complications have been observed as a result of the use of I^{131} in the management of thyrotoxicosis. They are discussed briefly in the following paragraphs.

Acute thyroiditis: In some cases (the number varies with the various authors), tenderness of the gland appears within 3 to 4 days after the radioactive material is administered. This usually subsides in about 10 days, but it has been known to persist for several months.[2, 32]

X-ray sickness: Nausea, vomiting, malaise, and fever seen following roentgen ray therapy may occasionally appear in the course of treatment.[31, 32]

Thyroid crisis: It has been thought that this could occur if too little radioactive isotope is used. However, in experimental rats,[9] it has been shown that it is the largest dose of I^{131} which produces the greatest elevation of values for protein bound iodine in the serum, and hence, might be thought to cause exacerbation of the thyrotoxic state. The rise is probably due to release of breakdown products from the gland into the circulation. All investigators have reported a temporary increase in the thyrotoxicosis in some patients.

Exophthalmos: The tendency for malignant exophthalmos to occur is greatest after surgery and least marked following x-ray therapy,[21] while its incidence with I^{131} occupies an intermediate position.

Myxedema: The incidence of this complication has varied with different groups of observers throughout the country and has been transient in many instances. In some cases, desiccated thyroid is given for three months, and then it is no longed needed.[35] Its incidence increased with very large doses of the isotope, it is rarely seen when the thyroid uptake does not exceed 100 microcuries per estimated gram of tissue, but it has been observed when as little as 35 microcuries per gram of tissue were used.[34] Twenty to 25 per cent may become hypothyroid.[2, 31]

Cancer: As has been stated previously, the possibility that cancer may follow the beta radiation with I^{131} cannot be denied, although no cases have thus far been reported in which such an association has been considered likely. Because the possibility cannot yet be completely discounted,[4] the majority of investigators prefer to employ I^{131} only in patients of the older age group, although malignancies due to radiation are usually associated with chronic, low intensity exposure, and not brief radiation.[2]

Some of the advantages of radioiodine over other forms of treatment are: (1) the simplicity of the procedure; (2) lack of interference with the patients' routine of living; (3) absence of skin lesions seen following roentgen ray therapy; (4) no mortality due to this type of treatment;[1] and (5) the relative infrequency of toxic manifestations.[1]

The disadvantages include: (1) the necessity for trained personnel and expensive equipment; (2) special precautions for handling a radioactive material; (3) the possible influence of radioiodine upon the urinary tract;[21] and (4) difficulty in ascertaining the evenness with which the effect has been exerted throughout the thyroid gland.

SUMMARY

1. Radioactive iodine has become a valuable agent in the study, the differential diagnosis, and the treatment of thyroid disease.

2. The development of radioactive iodine treatment is dependent upon the work of Marine, who showed the affinity of the thyroid gland for iodine, Fermi who developed radioactive iodine, and the early work of Hamilton, Lawrence, Hertz, and others.

3. I^{131} has been used in the study of the metabolism of iodine.

4. The rationale of the use of radioactive iodine in thyroid disease is based on physiological experiments and anatomical (or pathological) facts. These are considered.

5. With selection of patients and proper therapy, good results may be expected. The proper selection, treatment and expected results are discussed, as are the contraindications and complications of this type of therapy.

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This will be followed by a three-day course on PUBLIC HEALTH AND PREVENTIVE MEDICINE, **March 17, 18 & 19,** with the Wednesday program arranged jointly for the two groups attending these courses.

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March 11 & 12, 1954

A two-day session in actual microscopic examination of peripheral blood and bone marrow preparations of various hematologic diseases. Also, colored photographic slides will be projected for study. Ample time will be allotted for question and answer sessions.

Faculty:

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Kurt R. Reissmann, M.D., Associate Professor of Medicine. And Assistants.

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THE KANSAS PRESS LOOKS AT MEDICINE

Editor's Note. In this section the JOURNAL *reproduces editorials relating to medicine which have appeared in the lay press. An effort is made to include both favorable and unfavorable comments, and the Editorial Board in no instance assumes responsibility for the opinions expressed.*

APPENDIX FOR SALE

Medicine is a profession, not a merchandising service. Thus doctors have traditionally made varying charges for essentially similar operations and treatments according to such factors as individual prestige, wealth or poverty of the patients, and specific difficulties or complications that may be encountered.

For that reason, many of them are looking with more and more of a jaundiced eye at the medical and surgical insurance plans which set maximum payoffs for specified services.

It is true that these are only agreements between insurance companies and insured. There is nothing to keep doctors from charging their own price and collecting the difference from the patient. And they do it in thousands of instances.

But the pressure to conform to insurance schedules is becoming almost irresistible in places like Akron, Ohio, where group insurance plans cover the entire rubber industry and therefore almost the entire working population.

If the schedule says $100 for an appendix operation, and if the majority of doctors are charging $100 to middle-class patients, the upper crust patients can see no reason why they should pay more.

Nor can the man who has "complications." The doctor has plenty of easy, normal cases, hasn't he? Why can't he take a few tough ones in stride without tacking on an extra charge?

He doesn't have the credit losses he used to, does he? Then why should he charge the rich more to make up for a mythical "poor" which he no longer has?

These are hard questions to answer. Yet there is validity in the complaint that in areas where group insurance is almost universal, doctors tend to be forced into a straightjacket that bears a startling resemblance to socialized medicine.

In fact, it can almost be charged that it is social-

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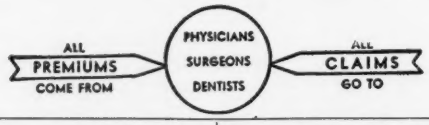

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Operating Room in Hospital	10.00	20.00	30.00	40.00
Anesthetic in Hospital	10.00	20.00	30.00	40.00
X-Ray in Hospital	10.00	20.00	30.00	40.00
Medicines in Hospital	10.00	20.00	30.00	40.00
Ambulance to or from Hospital	10.00	20.00	30.00	40.00
COSTS (Quarterly)				
Adult	2.50	5.00	7.50	10.00
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Child over age 19	2.50	5.00	7.50	10.00

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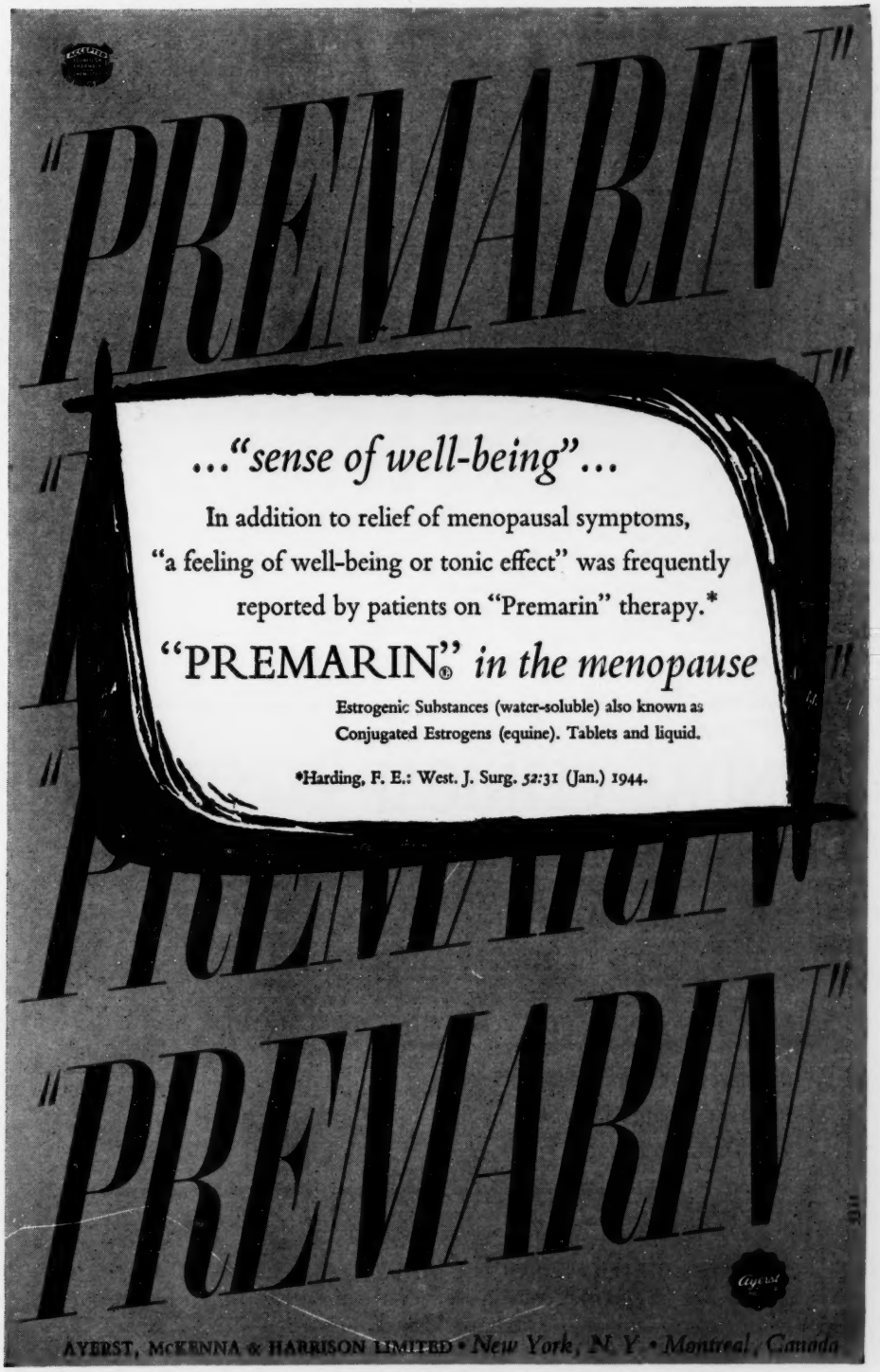
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*Harding, F. E.: West. J. Surg. 52:31 (Jan.) 1944.
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ized medicine. Because when its cost is paid as a deductible expense by a corporation, the government stands half of it in tax loss.

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The horse and buggy days of doctoring are gone. And some of the horse and buggy methods of charging for a doctor's service are just going to have to go along with them.—*Iola Register, November 16, 1953.*

Gift From Tuberculosis Association

A gift of $10,000 a year for five years to the new chair of thoracic diseases at the University of Kansas School of Medicine was announced recently by the Kansas State Tuberculosis Association. Each annual contribution will represent proceeds from the sale of 1,000,000 Christmas seals.

Dr. Martin James FitzPatrick, who joined the faculty of the medical school two months ago as assistant professor of medicine, will direct the work of the new chair. He is a diplomate of the American Board of Internal Medicine, an associate fellow of the American College of Chest Physicians, and a member of the American Trudeau Society and of the American Federation for Clinical Research.

The New England Medical Journal recently reported that one doctor who carefully surveyed his mail over a 12-months period, May 1, 1952, to April 30, 1953, received a total of 3,305 pieces of direct mail advertising. Of this total, 2,883 pieces were from pharmaceutical manufacturers. Products used in the treatment of cardiovascular disease headed the list, with antibiotics and sulfonamides next.

Demand for A.M.A. radio transcriptions on common health subjects hit an all-time high during 1953, according to a recent release from association headquarters. More than 700 sets were distributed for broadcasting over local radio stations in all parts of the country. Assisting the Bureau of Health Education in distributing these health education platters and promotional material are 14 distribution centers set up by state medical societies.

The transcriptions are provided without charge by the A.M.A. as a public service. Requests from groups not associated with the medical profession, such as universities and high schools, are always cleared through the local medical society.

The 1954 meeting of the American Medical Association will be held at San Francisco, June 21-25.

ANNUAL CLINICAL CONFERENCE

CHICAGO MEDICAL SOCIETY

March 2, 3, 4, 5, 1954

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*Stead, E. A., Jr., in Cecil, R. L., and Loeb, R. F.: Textbook of Medicine, ed. 8, Philadelphia, W. B. Saunders Co., 1951, p. 1065.

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BOOK REVIEWS

Respiratory Diseases and Allergy. By Josef S. Smul. Published by Medical Library Company, New York. 80 pages. Price $2.75.

In this book the author attempts to cover some of the major diseases of the organs comprising the respiratory system in the incredible space of 52 pages of text. In his preface he claims to be able to do this in a complete manner, but never quite achieves this end. As is usually the case in this type of endeavor, a "new approach" is heralded as the magic formula which attempts to reduce the multiple problems to fit the limited confines of this book.

Dr. Smul is able to break the complex interplay of infection and allergy into six compartments. He mentions the word allergy repeatedly but fails to define it. He coins new words which fail to contribute to a better understanding of fundamental problems. There is only one reference noted in the entire text. There is no mention of the extensive experimental work that has been done in this broad field. Clinical material has not been used in a recognizable form.

The author should have indicated the level of medical knowledge he was aiming at, in writing this book. The factual inaccuracies and uncompromising tone of presentation of the subject matter limit the scope and usefulness of this book to the student or to the practitioner.—*M.J. FP.*

Diseases of Women. By R. J. Crossen. Published by C. V. Mosby Company, St. Louis. 935 pages, 940 illustrations, 41 colored illustrations. Price $18.50.

This book is a revised edition of a long established text and reference book in which medical gynecological problems are clearly and systematically covered.

The chapter arrangement and method of material presentation are essentially unchanged. Many of the chapters have only minor revisions; more extensive changes consist mainly of adding or revising sections within chapters.

Some of the phases of gynecology new in this text are sections on the controversial subjects of intraepethelial carcinoma and factors predisposing to carcinoma; the psychosomatic aspect of puberty and adolescence as a method of preventing later psychosomatic problems; and embryology of the ovary as it is related to oogenesis and development of various ovarian structures.

The section on radiation therapy of cervical carcinoma is completely rewritten by Dr. A. N. Arneson.

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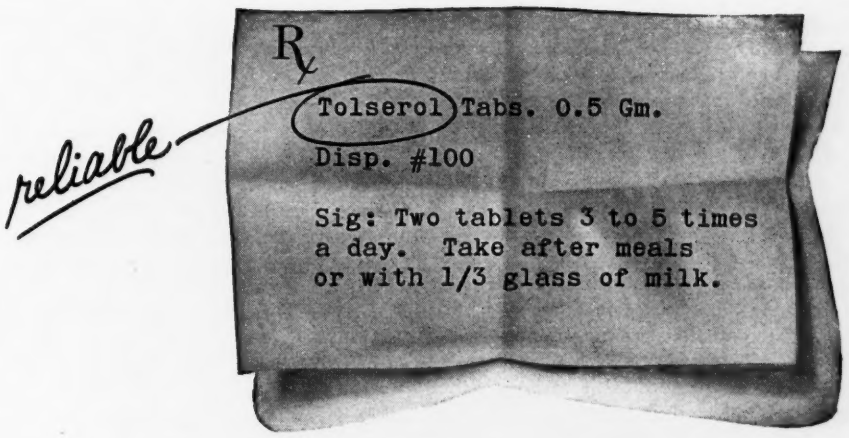

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SQUIBB

In this section basic physics and its practical application in radiation therapy is clearly and concisely explained.

Dr. Willard Allen not only wrote an excellent section on "Endocrine Relations Concerned in the Ovarian Cycle," but also in other sections made valuable additions on the therapy of endocrine disturbances.

The usual excellent chapter on "Gynecological Examination and Diagnosis" is included. In this chapter the steps and techniques of taking a proper gynecological history and making an adequate examination are outlined in detail. Also in this chapter special examinations and new diagnostic aids such as culdoscopy, culdocentesis, vaginal smears, tests for ovulation, and determining the exact day of ovulation and new pregnancy tests are added.

The chapter on "The Diseases of the Ovary and Parovarium" is revised with particular emphasis placed on grouping those related in origin and presenting the newer concepts concerning the origin of ovarian tumors.

This edition of an excellent text maintains the same high standards that have been associated with it since the first edition appeared in 1907 by Dr. H. S. Crossen.—*W.J.S.*

Patronize JOURNAL advertisers.

BOOKS NEEDED IN ISRAEL

An appeal for medical books published since 1940 was issued recently in an effort to relieve a shortage of such volumes in hospitals, universities, and offices of physicians in Israel. The United States State Department, sponsor of the project, will provide funds for shipment of the volumes from New York to Israel. Donors are asked to send contributions to Books for Israel, 115 King Street, New York 1, New York.

The past 50 years have been momentous in the fight against tuberculosis. At the outset it was found that the control of the disease was the problem both of the physician and the layman. . . . The achievements have been substantial. Our horizon has been raised to new and challenging vistas beyond. Despite our gains, we still are dealing with a serious killing disease that has sacrificed countless lives that should have been preserved for productive effort. So far as we can see at this time, tuberculosis will be with us for many decades to come. But we are drawing nearer to a complete understanding of its cause and control, which is beyond the tubercle bacillus alone. When we learn how to maintain our factors of resistance at a level that will keep the infection dormant, we may achieve as practical a result as

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complete eradication of the tubercle bacillus and the clinical manifestations of the germs in our bodies.—*H. R. Edwards, M.D., American Review of Tuberculosis, March, 1952.*

"We speak of medicine as both a science and an art, and surely these two aspects are complementary. Science is analytic, explaining, or seeking to explain, the whole in terms of the part; art is intuitive, and sees in the whole something more than can be explained as the sum of its parts. Alike in its comprehensiveness, its balance and its insight, the medicine which we have inherited from the great masters of the past in unique; and if we remain true to its philosophy we need have no fears for its future."—*Sir Russell Brain in the Lancet, quoted in Westchester Medical Bulletin, July, 1953.*

K.M.S. annual meeting, May 2-6, 1954, Topeka.

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